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FURTHER STUDIES OF THE POLLEN CONTENT OF THE MELBOURNE AIR.

By M. M. SHARWOOD, B.Sc. (Melbourne).

(From the Baker Medical Research Institute, Alfred Hospital, Melbourne.)

WHEN a survey of the pollen in the atmosphere during the previous hay fever season was being made,[1] it was felt that the technique was not very accurate and that the counts were not made frequently enough. It was decided, therefore, to repeat the whole survey and to make counts every day. Unfortunately the wind vane described below was not made available for use until October, so that the old method had to be used during the season when the trees were pollinating, that is, from August to October. This period corresponds also to the early portion of the season, when the grasses pollinate. The graph of the occurrence of the tree pollens is included in this paper, however, for comparison with that of the previous year.

Apparatus and Method.

Figure I is a photograph of the modified wind-vane used for collecting the pollen. To one end of the horizontal bar of the wind-vane an oblong shelter was attached, within which a microscopic slide three inches by one inch was placed at an angle of 45° to the horizontal. The wind could blow freely through this shelter, yet the slide was protected from rain. The other end of the bar was fitted with an aluminium tail. The apparatus was erected on a flat roof, thirty-nine feet high, in the Alfred Hospital grounds. At 9 a.m. each day a fresh microscopic slide coated with glycerine was placed in position on the vane and fastened with adhesive tape. From time to time the glycerine dried excessively, when more was applied to enable the

count to be done. Since the pollen grains collected were so much more numerous than those of the previous year, a coverslip three-quarters of an inch square was used instead of one seven-eighths of an inch square. On November 29 the slide was blown out of its position by the force of the wind, which was still so strong next day that another was not exposed. On three other days, namely, November 7 and 25, and January 31, no pollen was found on the slide, which appeared to have been washed by the rain. Apart from these incidents the method was satisfactory.

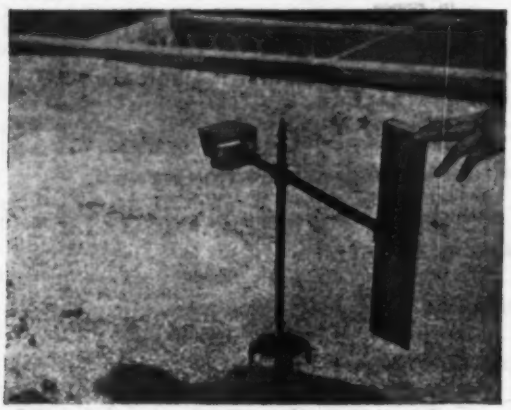

FIGURE I.
Modified wind vane.

Results.

As in the previous paper,[1] Charts I, II and III illustrate the influence of weather upon the amount of pollen present in the atmosphere.

The relationship of the pollen count to the rainfall is illustrated in Chart I. Dry periods varying from four to eight days occurred during October, November and December, and still longer periods during January, February and March. During all these dry periods the pollen counts were fairly high, while during the very wet periods, they fell practically to zero, but there were days, namely, November 24, December 26 and January 12, when several points of rain were recorded, yet the pollen counts were high. These were all warm, sultry days, with a small rainfall in the evening.

From a consideration of the two graphs in Chart II, a fairly close inverse relationship between the pollen content of the atmosphere and its relative humidity is noticed. For example, on November 12 and 23 and December 13 the pollen counts were high and the humidity was abnormally low. However, from December 18 to 24, there is a general upward trend in the two graphs, but the relative humidity remained below 50% and was probably insufficient to cause the pollen to fall out of the atmosphere. On January 22 the relative humidity was at its lowest and the pollen count was falling. This is so unusual that one is led to suspect some inadvertent error in technique. No experiments have been done in which dry pollen has been exposed for long periods to atmospheres having different relative humidities, the pollen being weighed before and after the exposure, so we have no data giving the actual increase of weight of pollen under these conditions. It is hoped to carry out these investigations at a later date.

Chart III illustrates the relationship of the northerly winds to the pollen counts, the amounts of wind being obtained by the same method as previously.[1] There is some correlation between the two graphs, but less than in the previous season. The highest peaks in the pollen graph again coincide with those in the wind graph, but there were days of strong northerly wind when the pollen counts were low. The counts for both November 7 and January 8 were very probably interfered with by the rain. On December 17 a large quantity of red dust was found on the slide, which may have either obscured the pollen or else prevented it from adhering to the slide. In March northerly winds were prevalent and the pollen counts were very low, but these results correspond to those obtained last season and were due doubtless to the dying off of the grasses.

The quantities of grass and plantain pollens found in the air are given in Chart IV. The total count made each day was, of course, much higher than that of the previous year, but in addition the proportion of plantain to grass pollen was much greater. In this season also the main flowering period of the grasses finished more definitely at the beginning of January. The plantain had its maximum flowering period in December and was still quite prevalent in January, whereas in the previous year it had a short maximum flowering period from the end of December to the beginning of January. The other weeds, sorrel, dock and chenopodium, were present in about the same proportions as in the previous year, and since the contents of sorrel and dock pollen vary with that of plantain, and the amount of chenopodium pollen is very small, these three were not included in the chart.

Patients' Symptoms.

During the season when the counts were being made, ten of the hay fever patients receiving treatment at the allergy clinic of the Alfred Hospital were asked to keep daily records of their attacks. Two only of these ten patients kept very complete records, and it was thought worth while to graph the frequency of their attacks against the amount of north wind and the pollen content of the atmosphere (see Chart III).

Patient A first attended the clinic on October 6, 1933. For two months previous to that she had suffered from nasal obstruction and slight asthma. Examination of the nose and throat revealed an allergic nasal mucosa and a healthy throat. Skin tests, made in October, 1933, revealed a strong positive reaction to all the grasses, a small reaction to sorrel, but none to plantain or any of the *Compositæ*. Several times during the season she received treatment for her nose, the middle turbinate being cauterized with trichloracetic acid, which is reported to have given her

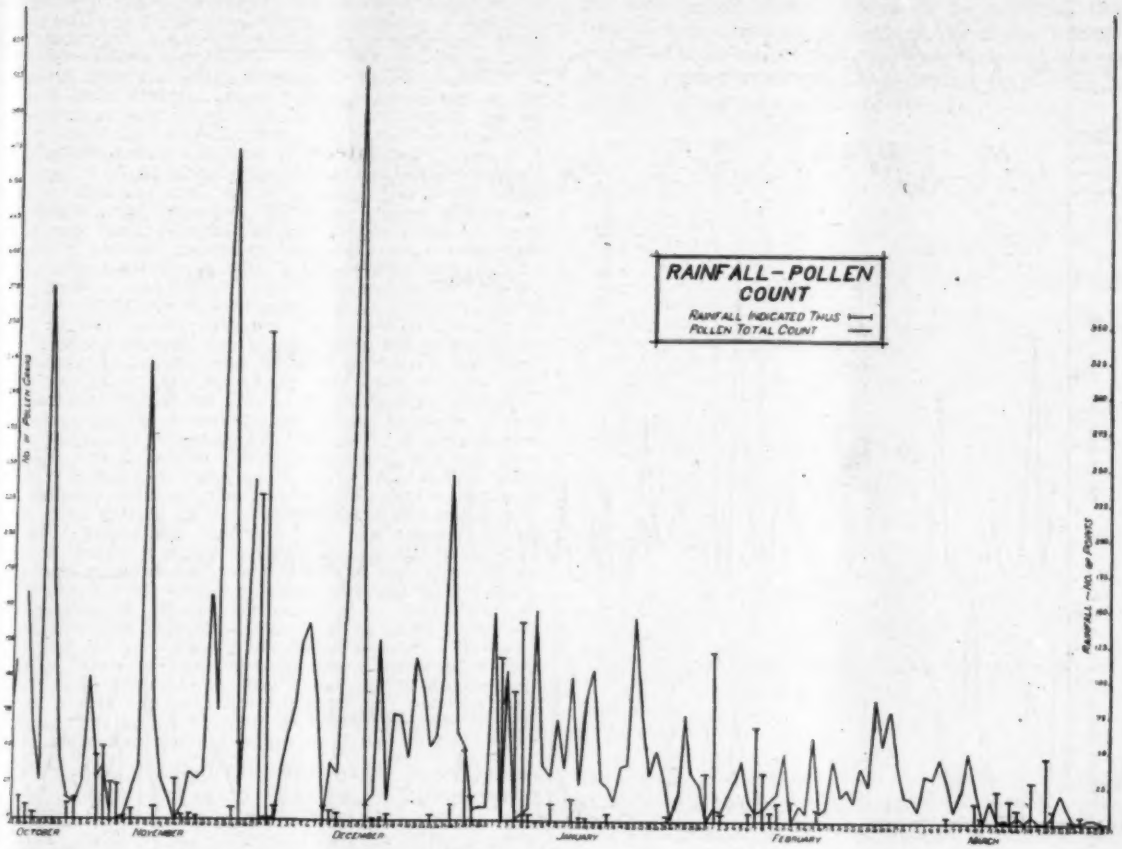

CHART I.

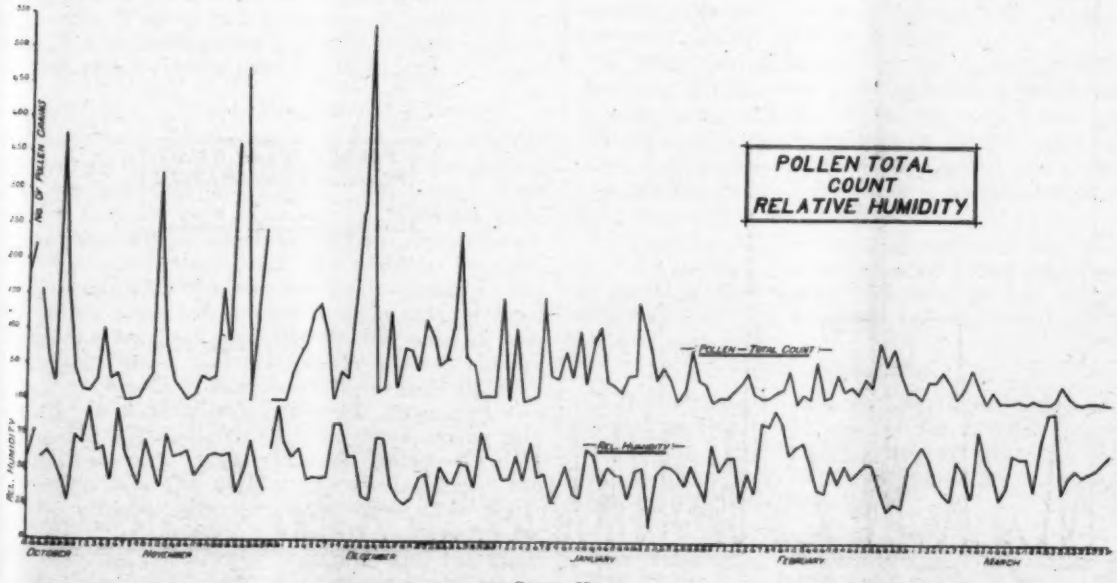

CHART II.

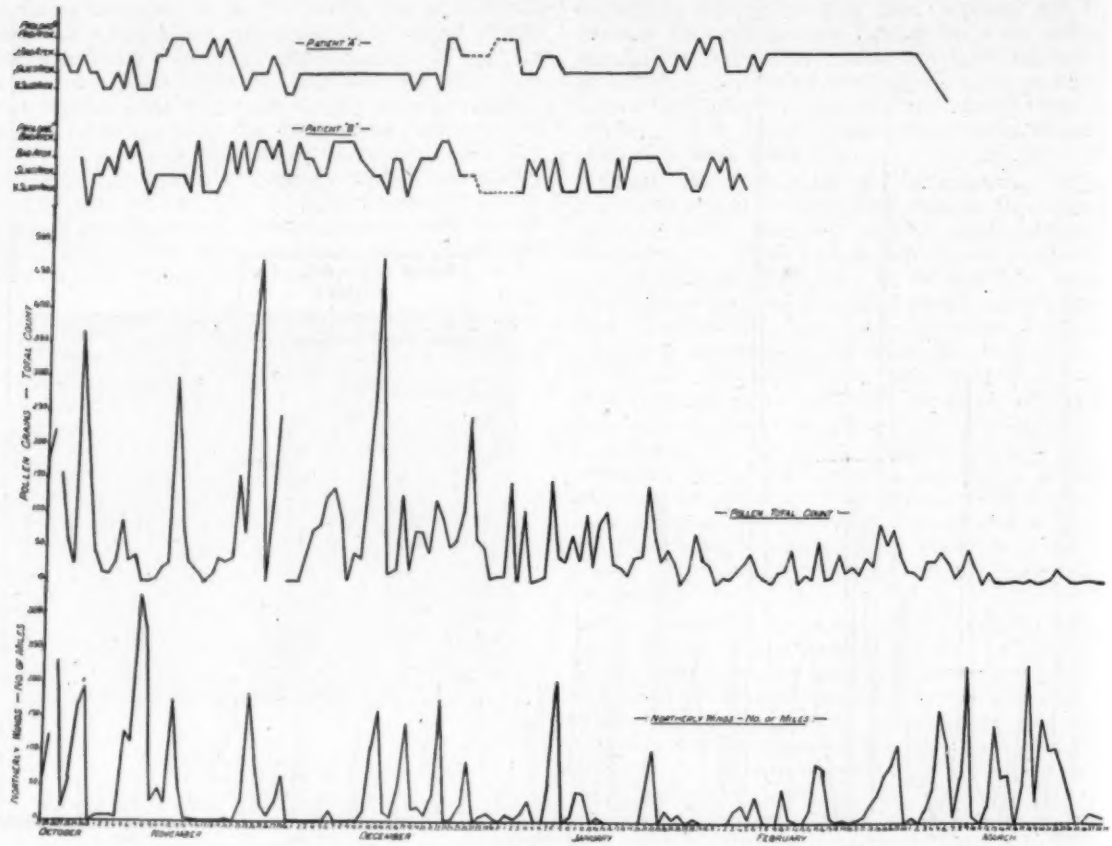

CHART III.

Correlation of the variations in the pollen content of the atmosphere with variations in the clinical conditions of two patients. (The dotted lines indicate the changes of residence of the two patients.)

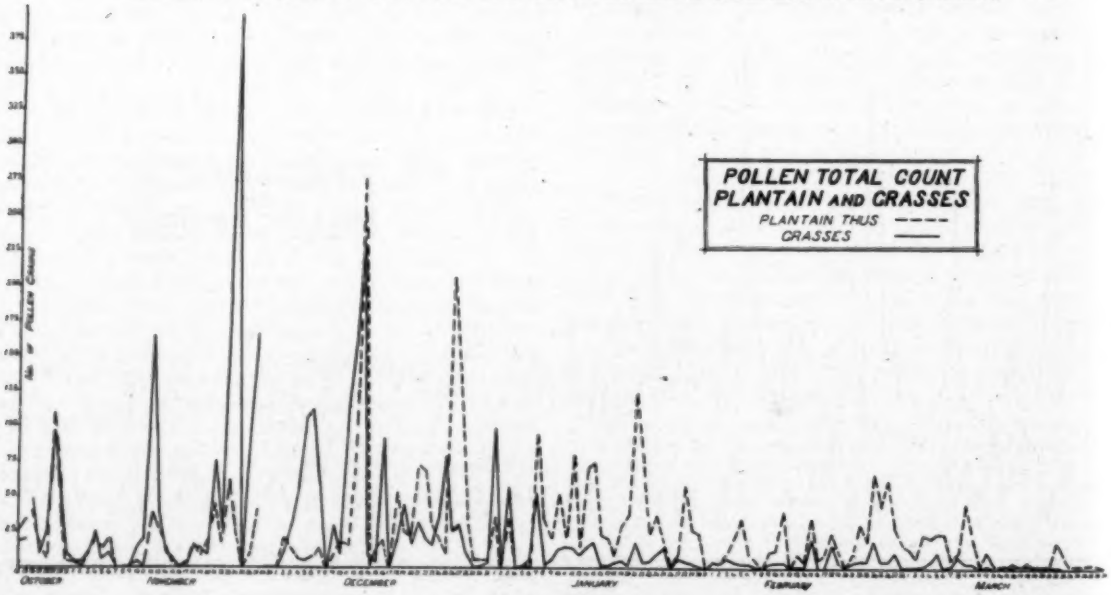

CHART IV.

considerable relief. She had been receiving injections of pollen extracts for a year before this season commenced and these were continued during the period over which the observations were made. She was reported to have improved with each batch of extract. These extracts were derived from different grass pollens. The graph of the frequency of her attacks shows no relationship to the pollen count, nor to the northerly winds, but her symptoms were aggravated by a holiday in the country.

Patient B attended the clinic for the first time on October 2, 1934. She had a history of perennial hay fever, which was worse from October to February, also of indigestion and severe headache. She had undergone a frontal sinus operation, but had had no injections nor other treatment. X ray examination revealed a slight mucosal hypertrophy of both antra. Examination of the nose and throat revealed infected tonsils and allergic nasal mucosa. She was tested with all the common allergens, and reacted strongly to all the grass pollens, also to capeweed, dahlia, sunflower and plantain pollens, and slightly to sorrel and dock pollens. She was first inoculated on October 26, 1934, with an extract of the mixed pollens of prairie, rye, cocksfoot, Yorkshire fog and timothy grass, and of plantain. These injections ceased on January 31, 1935, when it was reported that she was no better. Five months later she still gave strong reactions to the grasses, and began having more injections in private. The treatment, therefore, appeared to have no bearing on her attacks during the season considered. The graph indicates that the periods of bad attacks usually followed the days when the pollen counts rose very considerably, but did not coincide with them. There was no bad attack following the high pollen count of December 26, but she had moved from Canterbury, an inland suburb of Melbourne, to the seaside, which apparently accounted for her escaping the causative agent. However, the bad attack recorded on November 19 must have been due to some factor other than pollen in the atmosphere, as the counts had been low for four days prior to that. Whether this attack was due to her hypersensitive state or to some other allergen, we are not in a position to state.

These two graphs, therefore, show a difference between the reaction to external conditions of a fully treated and of a partially treated patient. Yet the symptoms of the partially treated patient were not entirely related to the amount of pollen in the atmosphere. However, physicians with wide experience of such cases do not consider that the symptoms of these two patients are typical; it is usual for pollen-sensitive patients to have attacks directly the northerly winds arise.

Tree Pollens.

Chart V records the results of an analysis of the tree pollens in the atmosphere, made by the method used for the previous season. The slides were exposed only on week-days, so that there are gaps in the observations which make them less satisfactory, but comparison with the previous season's graph shows that the two are fairly similar. The elm pollen appeared in even greater amount than during the previous year, but for the same period. The pine, cypress and oak pollens showed similar flowering periods. There is a considerable liability to error in distinguishing between plane and elm pollen, but it is believed that the error during this season was very small. The elm pollen grain is polyhedral in shape, with a very thick extine. The five germ pores are not easily seen. The plane pollen grain is smaller and has three germ pores, but these are not always noticeable and the thick extine gives it an appearance somewhat similar to the elm pollen grain. There is no evidence that tree pollens cause hay fever in Victoria, but the graph of their occurrence has been included in both papers in order to give as complete a picture as possible of pollen content of the air.

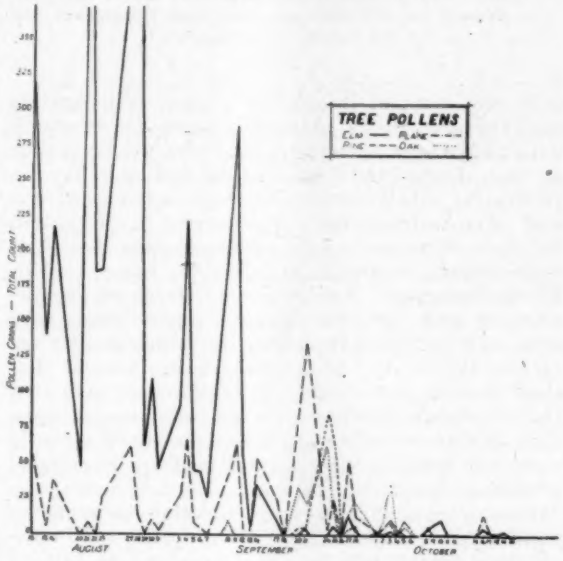

CHART V.

Summary.

By using a more accurate method for collecting pollen grains from the atmosphere it was found:

1. That the frequency of the occurrence of the commoner types of pollen was fairly similar to that of the previous season.

2. That this season the percentage of relative humidity showed a closer correlation with the pollen content of the air than last season.

3. That the relationship of the rainfall and the northerly winds to the pollen content of the air was in accordance with that of the previous season.

4. That a patient who had received nasal treatment and injections of pollen extracts for a year was unaffected by the amount of pollen present in the air or by the northerly winds.

5. That another patient suffering from hay fever and not having received sufficient pollen injections to give her any benefit, had most of her attacks following the days when the pollen content of the air was high.

Acknowledgements.

I should like to thank Dr. Charles Sutherland for suggesting this work, and for the interest he has taken in it. Mr. Barkley, of the Weather Bureau, has also been very helpful in supplying apparatus and meteorological data.

Reference.

(1) M. M. Sharwood: "The Pollen Content of the Melbourne Air during the Hay Fever Season of August, 1933, to March, 1934", THE MEDICAL JOURNAL OF AUSTRALIA, March 16, 1935, page 326.

INFESTATION OF MAN WITH TRICHOSTRONGYLUS COLUBRIFORMIS FROM SHEEP.

By I. CLUNIES ROSS.

(From the McMaster Animal Health Laboratory, Council for Scientific and Industrial Research, at the University of Sydney.)

A FEW cases of infestation of man with species of *Trichostrongylus*, normally parasites of ruminants, have been reported in the literature from time to time. Looss (1905) records the finding of *Trichostrongylus colubriformis*, *Trichostrongylus vitrinus* and *Trichostrongylus probolurus* in Egyptian fellahs. *Trichostrongylus colubriformis* has also been found in India by Clayton Lane (1913). *Trichostrongylus colubriformis*, *Trichostrongylus vitrinus* and *Trichostrongylus skrjabinema* have also been recorded from man in Armenia (Kalantarjan, 1934). It has generally been assumed that these species are accidental parasites of man and that cross infection has occurred from normal hosts, such as sheep or other ruminants, but until recently there has been no experimental evidence in support of this supposition. However, Heydon and Green (1931) were able to set up infection in goats by administering infective *Trichostrongylus* spp. larvæ obtained from human fæcal cultures, and on autopsy numerous specimens of *Trichostrongylus colubriformis* and a smaller number of *Trichostrongylus extenuatus* and *Hæmonchus contortus* were recovered. There appears no doubt that the *Trichostrongylus colubriformis* was derived from the larvæ administered; but Heydon and Green were unable to exclude all doubt as to whether the few specimens of *Trichostrongylus extenuatus* and *Hæmonchus contortus* might not have been acquired as natural infestations by the goats. *Hæmonchus contortus* larvæ had not been noticed in the cultures.

Recently the opportunity occurred of investigating the possibility of cross infection with *Trichostrongylus* spp. from man to the sheep. Two laboratory attendants at the McMaster Animal Health Laboratory, who were associated with experimental work on the small Trichostrongyles of sheep which necessitated the frequent handling of infective *Trichostrongylus* spp. larvæ, were both found to be passing strongylid eggs in their fæces. On culture, *Trichostrongylus* spp. larvæ were recovered. It appeared probable that such infections had been set up by the ingestion of *Trichostrongylus* larvæ of ovine origin.

In the first instance 610 larvæ obtained by fæcal culture from attendant B were administered to lamb S.770, which, except for the first two weeks of life, when it had been kept in a concrete pen which was hosed out daily, had been reared in a cage on bottled cow's milk. A four-weeks-old lamb which had been reared under similar conditions was run with it as a control. Daily examinations of the fæces of these lambs for one week before exposure to infection failed to reveal the presence of any parasites, with the exception of *Strongyloides* spp. Unfortunately, strongylid eggs appeared in the fæces of lamb S.770 ten days after exposure to infection and increased in number rather rapidly from day to day. In view of the period after experimental infection at which these eggs appeared being less than the minimum time required for the development of gravid female worms, it was concluded that a previous accidental infection had occurred. The lambs were therefore discarded. This serves to demonstrate the great difficulty experienced in guaranteeing absolute freedom from accidental parasitic infestations even in penned lambs.

Two lambs, S.840 and S.841, were then taken on the day they were born and reared in cages entirely apart from all other sheep, being fed on the bottle with cow's milk. The fæces of these lambs were examined three times a week, and over a period of three weeks they remained entirely free from all evidence of parasitism. As at this time the fæces of attendant A were free from all parasitic eggs, 210 infective *Trichostrongylus* larvæ derived from attendant B were administered to S.841. Fæcal examination of both lambs by direct centrifugal flotation was continued three times a week until the sixteenth day, and thereafter daily, seven to eight grammes of fæces being examined on each occasion. Eighteen days after the administration of infective larvæ, two strongylid eggs appeared in the fæces of S.841. On the following day, eight strongylid eggs were seen, the numbers increasing three days later to 250 eggs, and a week later to 400. The fæces of S.840 remained entirely "negative" throughout.

Lamb S.841 was destroyed twenty-six days after the administration of infective larvæ and the whole alimentary tract was examined in detail for evidence of parasitism. Parasites were absent from the abomasum, but from the small intestine five male *Trichostrongylus colubriformis* and twelve female *Trichostrongylus colubriformis* were recovered. No other parasites of any sort were found. The fæces of S.840 were examined at intervals for another fortnight, but remained entirely free from all eggs.

There appears to be little doubt in this case that the infestation in man was brought about with *Trichostrongylus colubriformis* of ovine origin, this being the commonest species employed in our experimental material, while experiments demonstrated that cross infection from man to sheep was easily effected. It is perhaps significant that the species involved in this instance was *Trichostrongylus colubriformis*, which has been reported more numerously from infestations in man than other ruminant species. That this species is less exacting in its host requirements than other *Trichostrongylus* spp. of sheep is suggested by its occurrence in a wide range of animals, while it is one of the two common ovine species of the genus *Trichostrongylus* which has been recorded in Australia from rabbits (Roberts, 1935).

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TRICHOMONAS VAGINALIS VAGINITIS AS A CAUSE OF PRURITUS VULVÆ.[1]

By BRIAN H. SWIFT, M.C., M.A., M.D. (Cantab.), F.R.C.S.E., F.R.A.C.S., M.C.O.G.,

Honorary Assistant Gynæcologist, Adelaide Hospital; Honorary Obstetrician, Queen's Maternity Home, Adelaide.

THE discharge caused by the infection of the vaginal epithelium with *Trichomonas vaginalis* has been found to be one of the commonest causes of *pruritus vulvæ,* and at the present time at least one or two new cases are being seen each week at the gynæcological out-patient clinic. However, it must be stated that the discharge caused by this organism does not always cause irritation of the vulva.

Literature.

Donné[(1)] in 1837 observed that a flagellate parasite was present in certain vaginal secretions, and since then numerous observers have found the organism in abnormal vaginal discharges.

The medical literature of Great Britain and Australia does not contain many papers on the subject, and it is only during the last decade that the condition has received much attention.

Andrews[(2)] published a paper in 1929, and described in detail the morphology and cultivation of the type of *Trichomonas vaginalis* found in England.

The first mention of infection of the vagina by *Trichomonas vaginalis* in Australia was a note of a case published in THE MEDICAL JOURNAL OF AUSTRALIA by Lethbridge[(3)] in 1931. Woodward,[(4)] Swift,[(5)] Matters,[(6)] Jacobs[(7)] and Graham and Collins[(8)] have published papers since 1931, but these authors confined their remarks to the clinical side and did not discuss the ætiology of the condition. Graham and Collins described their method of staining the parasite, but did not mention any method of cultivation, and, as far as I have been able to find, the literature does not contain a description of the Australian variety of *Trichomonas vaginalis.*

[1] Part of a work submitted for the degree of Doctor of Medicine in the University of Cambridge, May, 1936.

The American and Continental literature contains very numerous articles on this subject. A mass of literature on the signs, symptoms and treatment of the infection has accumulated, and a very full description of the flagellate as found in these countries with numerous methods of its cultivation has been published.

Incidence.

The number of cases of this infection seen during the last few years has certainly increased. It is difficult to say whether this is due to the fact that the signs of the infection have become more easily recognizable or whether the actual disease is spreading.

Gynæcologists have informed me that in the past they must have seen hundreds of cases of this infection, but that it was only during the last few years that they had realized what was the cause of the discharge. They were content in the past to make the ordinary dry film of the discharge, and so probably its real cause was missed.

It has been stated that the trichomonads were commonly found in the vaginal flora. Fifty consecutive gynæcological and obstetrical patients have been examined, and the vaginal discharge has been stained by the method which will be described later in the dissertation. Trichomonads were found in only four cases, or 8%, and of these fifty patients twelve had complained of discharge, and so trichomonads were present in 33% of the patients with discharge. (Cultivation of the discharges perhaps might have revealed a slightly greater percentage of trichomonads.)

The following is a list of the findings in fifty consecutive cases among ordinary routine gynæcological and obstetrical patients. Films were taken from the vaginal vault in each instance, and then were examined for the presence of *Trichomonas vaginalis.*

1. Numerous epithelial cells and Gram-negative rods. No pus cells, no gonococci nor *Trichomonas vaginalis.*
2. Numerous pus cells and *Trichomonas vaginalis* present.
3. Numerous pus and epithelial cells. No *Trichomonas vaginalis* nor microorganisms.
4. Numerous epithelial cells and large Gram-negative rods. No *Trichomonas vaginalis* nor pus cells.
5. Numerous pus cells and mixed Gram-negative rods. No *Trichomonas vaginalis.*
6. Numerous pus and epithelial cells and Gram-negative rods. No *Trichomonas vaginalis.*
7. Extremely numerous small Gram-negative rods with rounded ends, some pus and epithelial cells. No *Trichomonas vaginalis.*
8. Numerous epithelial and some pus cells. Numerous large Gram-negative rods. No *Trichomonas vaginalis.*
9. Numerous pus cells and Gram-negative rods and cocci. No *Trichomonas vaginalis.*
10. Numerous epithelial cells, few pus cells, numerous Gram-negative rods. No *Trichomonas vaginalis.*
11. Numerous small Gram-negative rods, some pus and epithelial cells. No *Trichomonas vaginalis.*
12. Extremely numerous small Gram-negative bacilli. Epithelial cells. No pus nor *Trichomonas vaginalis.*
13. Numerous pus cells and Gram-negative bacilli. No *Trichomonas vaginalis.*
14. Numerous epithelial cells and large Gram-negative rods present. No pus nor *Trichomonas vaginalis.*

15. Numerous epithelial cells and short Gram-negative rods present. No pus nor *Trichomonas vaginalis*.
16. Numerous epithelial cells, some pus cells and numerous small Gram-negative bacilli. No *Trichomonas vaginalis*.
17. Pus and epithelial cells, small Gram-negative bacilli. No *Trichomonas vaginalis*.
18. *Trichomonas vaginalis* and gonococci present. Some spermatozoa, numerous pus cells and Gram-negative organisms.
19. Numerous epithelial cells and Gram-negative bacilli. No *Trichomonas vaginalis* nor pus cells.
20. Numerous epithelial cells and large Gram-negative bacilli. No *Trichomonas vaginalis* nor pus cells.
21. Numerous pus cells and epithelial cells. Gram-negative organisms. No *Trichomonas vaginalis*.
22. Numerous pus and epithelial cells, Gram-negative organisms. No *Trichomonas vaginalis*.
23. Numerous epithelial cells and Gram-negative bacilli. Few pus cells. No *Trichomonas vaginalis*.
24. Numerous epithelial cells, pus cells and Gram-negative bacilli. No *Trichomonas vaginalis*.
25. Numerous pus cells and *Trichomonas vaginalis* present. Gonococci present.
26. Numerous pus cells. Gram-positive cocci. No *Trichomonas vaginalis*.
27. Numerous pus cells, epithelial cells and large Gram-negative rods. No *Trichomonas vaginalis*.
28. Numerous pus cells, epithelial cells and large Gram-negative rods. No *Trichomonas vaginalis*.
29. Numerous pus cells. No microorganisms nor *Trichomonas vaginalis*.
30. Many epithelial cells, few pus cells, Gram-negative rods. No *Trichomonas vaginalis*.
31. Many epithelial cells and Gram-negative rods. No pus cells nor *Trichomonas vaginalis*.
32. Pus and epithelial cells and masses of very small Gram-negative rods. No *Trichomonas vaginalis*.
33. Pus and epithelial cells and large Gram-negative rods. No *Trichomonas vaginalis*.
34. Numerous epithelial cells, few pus cells, small Gram-negative rods. No *Trichomonas vaginalis*.
35. Numerous epithelial cells, few pus cells, large Gram-negative rods. No *Trichomonas vaginalis*.
36. Epithelial cells, few Gram-negative rods. No *Trichomonas vaginalis*.
37. Epithelial and pus cells and some gonococci (intracellular cocci). No *Trichomonas vaginalis*.
38. Epithelial and pus cells and Gram-negative rods. No *Trichomonas vaginalis*.
39. Epithelial and pus cells and large Gram-negative rods. No *Trichomonas vaginalis*.
40. Pus cells. No microorganisms nor *Trichomonas vaginalis*.
41. Pus and epithelial cells and Gram-negative rods. No *Trichomonas vaginalis*.
42. Pus and epithelial cells and small Gram-negative rods. No *Trichomonas vaginalis*.
43. Pus and epithelial cells and small Gram-negative rods. No *Trichomonas vaginalis*.
44. Numerous epithelial cells. No pus cells. Large Gram-negative rods. No *Trichomonas vaginalis*.
45. Numerous epithelial cells, few pus cells and Gram-negative rods. *Trichomonas vaginalis* present.
46. Numerous pus and epithelial cells and Gram-negative rods. No *Trichomonas vaginalis*.
47. Pus and epithelial cells. Gram-negative rods. No *Trichomonas vaginalis*.
48. Pus and epithelial cells. Gram-negative rods. No *Trichomonas vaginalis*.
49. Pus and epithelial cells, mixed Gram-negative flora. No *Trichomonas vaginalis*.
50. Pus and epithelial cells, mixed Gram-negative flora. No *Trichomonas vaginalis*.

This number is small, but corresponds with the investigation of Graham and Collins[8] in Australia, who found that trichomonads were present in 30% of forty-six patients who had complained of leucorrhœa, whilst Andrews[2] found that 20% of women were infected. Glassman,[9] however, found that between 50% and 70% of all patients with leucorrhœa were infected with trichomonads. It can be seen from these figures that the presence of trichomonads is quite common in patients who have a vaginal discharge, and the percentage varies from 20 to 70.

Davis[10] was unable to find a single case of trichomonad infection in any normal woman who had no vaginal discharge. However, the percentage of infected cases appears to increase tremendously when the women are pregnant. Bernstine[11] found that in a series of 1,250 pregnant women, 10·56% were infected, whilst Bland, Goldstein and Wenrich[12] found that 23% were infected. Glassman[9] examined 309 pregnant women and found 20·7% infected and Cornell, Goodman and Matthies[13] found only 7·6% in 500 pregnant women.

The percentage of pregnant women who are infected varied from 7·6 to 23. Unfortunately, these authors did not state whether the pregnant women had complained of discharge or not. The statistics of pregnant women and non-pregnant women cannot be compared unless women without and with discharge are classed as one group, when the percentage of infection in all non-pregnant women would be very small.

An eroded cervix was the commonest cause of discharge in this investigation, but trichomonad infection was also very frequent. I am unable to quote a large series of cases, and can only state that very many more cases of this infection are now being seen. The youngest patient in this series was a girl of five years, and the oldest was a woman of seventy-two. There have been quite a number of infections in girls from the age of fourteen onwards and a large number were in virgins.

It is seen, therefore, from these figures, that infection by the *Trichomonas vaginalis* is a common cause of discharge, and that pregnant women are frequently infected, and it has been found that when trichomonads are present the woman complains of marked irritation.

Pathogenicity.

There is still some divergence of opinion as to whether the *Trichomonas vaginalis* is really the sole cause of the vaginitis or only one of many causes.

Hesseltine[14] stated in 1933 that the pathogenicity of *Trichomonas vaginalis* was still unproved. He also stated that the trichomonads failed to grow in the absence of bacteria, and that the trichomonad was a scavenger and fed on the bacteria. However, it was found in this investigation that the trichomonads grew better when the bacterial growth became lessened.

Hibbert[15] found that trichomonads were present in the vaginal secretions of women for a long time without causing an acute vaginitis. He also stated that a short chain streptococcus was present in a large number of cases, and that this streptococcus was capable of producing an active vaginitis when not associated with *Trichomonas vaginalis*. He con-

cluded, therefore, that the parasite was not the cause of the vaginitis. Streptococci, however, have been found in association with trichomonads in very few cases in this series of over one hundred.

Cornell, Goodman and Matthies[13] said that women could harbour the trichomonads without the production of any symptoms except an occasional increase in the vaginal discharge. This statement does not coincide with the finding in this investigation.

The number of gynæcologists who consider that the *Trichomonas vaginalis* is the cause of the vaginitis is increasing. There appears to be no doubt that the trichomonad does cause a very definite and typical discharge, and that a clinical cure is obtained only when the therapy has succeeded in eliminating the flagellates.

Ætiology and Infection.

A search through the literature did not throw any light on the source of the infection. Numerous theories have been suggested.

The theory of infection from the anus and rectum had been suggested because it had been found that numerous patients who were clinically cured of the vaginal infection became densely infected again after the next menstrual period. The wearing of a vulval pad during menstruation might have been the cause of the spread of infection from the anus to the vulva by a process of direct contact, the pad moving its position when the woman moved, or by a process of permeation in the moisture in the pad after it had become soaked in menstrual blood. However, during this investigation, it was found impossible to cultivate trichomonads from the fæces of infected patients.

There is definite evidence that a flare-up of the infection does take place after a period, and also that, if the treatment be continued during the period, this flare-up did not occur.

It has been found that after a menstrual period the trichomonads present in the vagina are very much smaller and much more actively motile than at other times and that they resemble closely *Trichomonas hominis*. This fact has no doubt been the reason for the above mentioned theory of infection from the anus. However, the reason for this flare-up after a menstrual period will be discussed later.

Another theory, which was rather attractive, was that the infection had been conveyed to the vulva by the fingers. Quite a number of my patients had given the history of having been touched by the fingers of another person. This theory could not be feasible unless the dried pus was still infective. In an attempt to prove this theory the following experiment was carried out to try to prove that the parasite was very resistant to drying:

Small drops of pus from the vagina of an infected woman were placed in a row on a piece of glass and allowed to dry in the air and in a dull light. After varying intervals of time each dried drop was mixed with warmed normal saline solution to which had been added some "Folliculin" solution. This mixture was then inoculated into a culture medium. Trichomonads were found alive and growing in culture even after the drop of pus had been allowed to dry for three hours. ("Folliculin" solution had been added to the normal saline solution so as to make a solution which approximated more nearly to the normal vaginal secretions.)

Rodecurt[16] found that trichomonads survived several freezing processes and drying. The above experiment was done, however, before Rodecurt's paper was seen.

The result of this experiment, namely, that drying did not kill the trichomonads and that dried pus was still infective, is most upsetting. Dried pus on the finger would infect a suitable place such as the vulva and the woman would become infected.

No case of urethritis in the male was encountered during this investigation, although regular intercourse had taken place between the males and their infected wives. In several cases it was possible to obtain samples of the prostatic secretion and this did not contain trichomonads. It must be concluded that the male urethra is not a place in which trichomonads will thrive. However, Riba[17] found that trichomonads were present in nine cases in the prostatic fluid of 3,000 men; whilst Allen, Jensen and Wood[18] state that they recovered trichomonads from the prostatic fluid of six husbands whose wives were infected. Riba[17] also quotes two cases of urethritis in the male due to trichomonads. Cornell, Goodman and Matthies[13] also quote a case of trichomonas urethritis in a male which occurred after connexion with an infected woman.

Trichomonas vaginalis was found to be present in the discharge obtained from the female urethra in one case only in my series, but there was not a definite urethritis in this instance.

It appears, therefore, that the urethra is very seldom a suitable place for the trichomonads to grow in or that the normal urethral discharge is not a suitable medium. The normal vaginal secretion, however, appears to be a suitable medium. It might, however, be suggested that the trichomonads, after entry into the vagina, may remain quiescent until the next menstrual period and only then start to multiply. This reproductive activity might be due to a stimulus present at menstruation; this stimulus could be the menstrual blood, but would more likely be the ovarian hormone which it contains. Winter[19] has suggested that the trichomonads have no effect on a healthy vaginal epithelium, but that some constitutional condition or trauma may unfortunately influence the vaginal walls so as to allow them to be infected by *Trichomonas vaginalis*.

It has been noticed that infected women who are past the menopause are more quickly cured than women who are still menstruating. This rapid cure may also be due to the lack of the ovarian hormone stimulus for reproduction. The male and female urethral discharges do not contain any ovarian hormone and hence trichomonads will not grow in these media.

Infection, therefore, becomes a real problem. A drop of infected pus on a lavatory seat is a big source of infection and can remain infective for several hours. Women must therefore be warned about touching lavatory seats or allowing paper to touch the seat before they wipe themselves. Greenhill[20] instructed his patients about the method of wiping the anus and advised that the movement be done from the vagina towards the sacrum. A much better advice can be given in the future and would be: "Do not let the paper touch the lavatory seat."

A very good example of the danger of the lavatory seat was the case of a patient of mine on whom I had done an abdominal operation and who went for a trip on a small steamer. She had noticed that the lavatory accommodation was very poor and dirty. On her return she developed a very bad irritation of her vulva with a profuse vaginal discharge.

On examination she was very badly infected with *Trichomonas vaginalis* and must have been infected on the boat. She informed me, however, that she never sat on lavatory seats, but only squatted; but she admitted that the paper might have touched the seat. As this woman could not have been infected in any other way and was not infected before she started the trip, the lavatory seat must be blamed for the infection.

The reply to the question of infection of others which is so often asked, is that from our experience the woman is infective to other women only.

An infected woman must be told that after she has micturated or defæcated, the seat should be well cleaned with an antiseptic. In fact, the same stringent precautions should be taken as with a Neisserian infection.

The ætiology of the infection still remains a mystery; but two points have arisen; first, that dried pus is still infective and remains infective for hours, and, secondly, that the ovarian hormone is necessary for the continual reproduction of trichomonads and so the vagina and vulva are presumably the only places which can be infected by *Trichomonas vaginalis*.

Pathology.

The introduction of *Trichomonas vaginalis* onto the vulva or into the vagina is followed by the formation of a greenish frothy discharge. On examination this discharge shows numerous pus cells with Dōderlein bacilli and epithelial cells and numerous trichomonads which in the moist preparations are actively motile.

The vaginal walls become inflamed and in places roughened and quite granular. The *caruncuIæ myrtiformes* are reddened and the cervix in some cases becomes covered with small red spots; but usually the cervix is not eroded.

This infection is sometimes also associated with the presence of a gonococcal infection. Bernstine[11] found in a series of pregnant women that 1·9% had both a trichomonad and a Neisserian infection. It has been stated by Jacobs[7] that it is unusual to find both infections present at the same time. During the routine examination of films at the Adelaide Hospital, the method of staining to be described later being used, it has been found on numerous occasions that trichomonads were present as well as gonococci (Figure IX). I think that in the past gonococci were noted, but no mention of trichomonads was made as they were not recognized.

Staining.

The following method of staining the parasites has been found most successful for routine use; it is a modification of Gram's stain and was suggested by Schmidt and Kamniker.[21]

A very important detail of technique is that an unflamed film of pus must be used. Any application of heat for fixing the film destroys the structure of the organism. The film therefore should be allowed to dry in the open air.

The presence of trichomonads in association with gonococci was not noticed in the past because the usual method of fixing the film over a flame was used. The structure of the trichomonads was destroyed, and they were then mistaken for pus cells.

Technique.—Oxalate methyl violet solution is poured over the dried film and allowed to remain for thirty seconds. This solution consists of:

Methyl violet V.B., saturated alcoholic solution, 5 cubic centimetres.
Ammonium oxalate, 1% aqueous solution, 95 cubic centimetres.

The stain is washed off with water and Gram's iodine is applied for sixty seconds. Decolorization by 90% alcohol is then carried out until the film shows no retention of violet colour.

All traces of spirit are removed by washing well and a strong carbol fuchsin solution is then used as a counter stain. Ewing and le Morne[22] recommend a diluted solution in their article. Ziehl's carbol fuchsin diluted with an equal amount of distilled water was found to be a satisfactory concentration.

Trichomonads were found to be very well stained by this method and after a little experience could be identified at once. The flagella in most cases were stained and could be easily seen by careful focusing. The vacuoles with ingested bacteria could be easily distinguished and in some cases the undulant membrane could be seen (Figures III, IV, V, VI, VII, VIII).

By this method trichomonads which have been taken on routine examination for gonococci can be detected in the ordinary films of discharge from the vagina and cervix.

Two important points must be remembered: (i) The film of pus must be allowed to dry in the air. (ii) The carbol fuchsin solution must be strong.

Cultivation.

The first attempts at cultivation of the trichomonads from vaginal pus were not very successful. The medium described by Hogue[23] was first tried and was prepared as follows. Ten to fifteen cubic

centimetres of sterile serum water were added to 100 cubic centimetres of 0·85% sodium chloride solution (after autoclavation). (In the description which was given by Hogue, sheep serum was used, but in these attempts human serum was substituted. The serum water was prepared by diluting one part of serum with three parts of distilled water, and this was sterilized by steaming at 100° C. on three successive days.)

Sterile test tubes containing 15 cubic centimetres of this serum-saline solution were inoculated, after being warmed to 37° C., with a small amount of pus from the vagina. The pus was taken either by means of a pipette or by using an ordinary sterile throat swab. It was found that the throat swab could be pushed quite easily into the vagina through the intact hymen of unmarried girls without causing much discomfort.

The surface of the medium was then covered with sterile paraffin oil and was incubated at 37° C.

No evidence of coagulation of the serum, as described by Hogue, was noted, but bacteria grew vigorously in every case. Trichomonads could be demonstrated in the sediment at the bottom of the tubes up to seventy-two hours after inoculation; but in no case could actual multiplication be noted, although the number of trichomonads per field in the sediment had greatly increased. This increase was thought to be due to concentration rather than to multiplication.

Reinoculation was then tried with this medium, but the excessive growth of bacteria appeared to swamp the growth of the trichomonads, and in no case was it successful.

The medium described by Lynch[24] was next tried, but with precisely similar results. Sterile human serum was added to urine from patients suffering from chronic nephritis and this medium was inoculated, but no growth of trichomonads was obtained, and no motile parasites could be demonstrated after more than twenty-four hours' cultivation at 37° C.

A medium which was described by Stein and Cope[25] was next tried. These authors have published several articles on *Trichomonas vaginalis* and its culture, which have been of very great assistance. A placenta agar slope was used, to which three to four cubic centimetres of sterile saline solution containing 10% of human serum was added. Inoculations were made into this medium, and the sediment at the base of the slopes was found to contain numerous actively motile trichomonads after forty-eight hours of cultivation. Subcultures were made, but the number of parasites unfortunately became less and less, whereas the amount of bacterial growth increased. This excessive growth of bacteria was noted in every case, and appeared to be the factor which was again inhibiting the growth of the trichomonads.

This medium was then tried with the addition of a solution of sodium citrate, as advocated by Andrews.[2] She had found that the addition of the citrate inhibited the growth of the bacteria. It was found, however, that the bacterial growth was only slightly inhibited in the early growth phase, no decrease could be noted after forty-eight hours of incubation, and subcultures would not grow.

A placenta juice filtrate was next tried instead of the serum citrate medium. The growth of the parasites appeared to be greater, but again subcultures became "negative" and the bacterial growth enormous.

Stein and Cope,[27] in their investigations on trichomonads, noted that an infected patient became very much worse after each menstrual period. They suggested that the presence of ovarian hormone in the menstrual blood, as noted by Frank and Goldberger,[28] was perhaps the reason for this increased activity of the parasites. Stein and Cope added 4% of "Progynon" to their saline mixture, and found that the activity and rate of growth of the organisms were greatly increased.

A serum saline citrate medium was next prepared and "Theelin" was added, so that the concentration was four rat units per cubic centimetre. This solution was added to test tubes with blood agar slopes. The medium was warmed to 37° C., inoculated and then incubated for thirty-six hours. The sediment at the bottom of the tubes showed very numerous parasites and a great increase in their numbers. Trichomonads in the process of division were sometimes seen and subcultures were done, but, as before, the bacterial overgrowth overwhelmed the protozoa.

It appears from this investigation that the female sex hormone plays some part in the life cycle of *Trichomonas vaginalis*, and that the excessive bacterial growth swamps the growth of trichomonads.

A recent publication by Wagner and Hees[29] describes a medium which was originally devised for the cultivation of *Entamœba hystolytica*. They found that trichomonads grew vigorously in this medium after incubation at 37° C. for forty-eight hours. This medium was modified slightly, and has been used with excellent results both as regards the cultivation of the trichomonads and the suppression of the bacterial growth.

A blood agar slant was used, and three to four cubic centimetres of the following solution was added: equal parts of 0·7% solution of sodium chloride and 1·0% solution of sodium citrate were mixed and adjusted to a pH of 7·4. "Folliculin" was added to make a concentration of four rat units per cubic centimetre, and acriflavine was then added to make a final concentration of 1 in 10,000. This concentration of acriflavine has an excellent inhibiting effect on bacterial growth and does not kill the trichomonads.

This medium is now being used with very good results for the routine cultivation of pus from women suspected of being infected.

Morphology.

The variety of *Trichomonas vaginalis* found in the British Isles has been described by Andrews,[2] who wrote:

Trichomonas vaginalis is a large, roughly spherical or oval body, measuring from 10 to 20·5 microns long by 8 to 19 microns in width. The undulating membrane only extends for one-half to two-thirds of the body length, and there is no free flagellum. There are four anterior flagella, one pair being slightly longer than the other. The length of the flagella varies from 6 to 19 microns. The nucleus is ovoid in shape and is situated near the anterior end. The axostyle only extends a short distance beyond the body, and the flagella and undulating membrane arise from a bunch of blepharoplasts anterior to the nucleus. The protoplasm is finely granular, and vacuoles may be present containing ingested bacteria.

However, in culture the trichomonas becomes smaller and the undulating membrane extends further down until there is a well-marked free flagellum and the axostyle extends some distance beyond the body.

The American variety has been described by Stein and Cope,[25] who stated:

Trichomonads may be pyriform, amœboid, spheroid or fusiform. Typically they are pear-shaped, rounded at the anterior end and pointed at the posterior end. Anteriorly four flagella arise from blepharoplastic granules. An undulating membrane arises on the anterior end and extends about two-thirds of the length of the cell wall. Close to the anterior end of the cell body there is a large spindle-shaped nucleus containing scattered chromatin granules and sometimes a karyosome. The body is finely granular and frequently contains numerous food vacuoles which may be filled with bacteria.

The size of the parasite was said to be 15 to 25 microns in length and 10 to 15 microns in width.

The German variety was well described by Schmidt and Kamniker,[21] who stated that:

The size of the parasite is very variable, but usually they are about the size of a polymorphonuclear leucocyte, but on occasions are two to three times as large. The shape is very variable, but usually they are spindle shaped.

They described the four flagella and the undulant membrane and the two spots from which they arise. They stated that:

The protoplasm of the trichomonads is not homogeneous, but one sees numerous small bodies of irregular size which sometimes contain ingested bacteria. These are described as vacuoles, but we do not consider them as true vacuoles, as they differ in number in different organisms and vary greatly in size.

It will be seen that the descriptions of the three varieties, namely, English, American and German, are practically similar.

Australian Variety.

Body of Trichomonad.—The size and shape of *Trichomonas vaginalis* have varied greatly in different specimens which have been examined. Forms have been found as small as seven microns in length and as large as twenty microns. The organisms are difficult to measure in the living state owing to the rapid amœboid movement.

The small form, which is ovoid in shape, is always to be found in the blood-stained fluid in the last days of the menstrual flow of an infected woman. These small forms are considered to be the young trichomonads and are extremely active, and are ever so much more active than the larger older types. When these small forms are observed under the high power or oil immersion objective the movements are so rapid that it is impossible to keep a single organism in the field under examination. In unstained specimens the young form appears as a highly refractile body. The protoplasm is clear and shows no evidence of vacuolation or ingested bacteria. In stained specimens the young forms stain evenly with the fuchsin and do not show a granular structure, nor can the nucleus be seen. (Figures I and II.)

The larger or older forms, which tend to be more circular in shape, move much more slowly, and the protoplasm appears to contain vacuoles which are full of ingested bacteria. When stained, the protoplasm appears granular (Figures III and IV), and the parasite has the power of amœboid movement (Figures V and VI).

It would appear that whilst multiplication is effected by binary fission, many parasites, probably the large granular forms, do not divide. The types which were seen in post-menstrual blood and in cultures and which showed two sets of flagella, were all of the small homogeneously staining type. (Figure VII.)

Flagella.—The flagella, which are slender threads of uniform thickness, were always four in number, and arose from a small body called a blepharoplast near the pointed end of the parasite, where a pronounced dip occurred in the outer membrane of the body. This small spot was situated to one side of the nucleus, and on the other side a similar spot was found from which the undulant membrane arose.

Undulant Membrane.—The undulant membrane, which is morphologically a flagellum, is continued closely along the ventral side, whose length is about half the length of the body of the parasite. The movements of the membrane are so rapid, especially in the small forms, that the number of undulations cannot be estimated. However, in the stained specimens there are usually eight undulations. (Figure VIII.)

The Nucleus.—The nucleus, which can usually only be made out in the older larger forms, is situated in the anterior end of the organism, and varies in size with the variation in size of the individual organism. It is almost in contact with the blepharoplastic spots. (Figure IV.)

The Axostyle.—The axostyle is a small rod of hyaline appearance, and appears to originate in the blepharoplastic spot and to extend posteriorly in an axial position. Its posterior end extends beyond the cell body as a pointed tip. The axostyle was very difficult to see in either the stained or moist preparations.

Movements of the Trichomonads.—The movements of the flagella are of a lashing, sweeping character, and some authorities say that they are used to sweep bacteria into a so-called stoma at the anterior end. (Figure X.) This stoma has not been noticed in the Australian variety. The organism as a whole changes its shape owing to its power of amœboid movement, whilst the undulating

membrane has a very rapid wave-like movement. (Figures V and VI.)

In moist coverslip preparations the trichomonads slowly lose their amœboid movements and become stationary. The flagella continue to lash vigorously and the undulating membrane still undulates. As the parasite dies the flagella cease movement, and the body assumes a circular shape. The movements of the undulating membrane are the last to stop, and often continue for some time after all other movement has ceased.

Symptoms.

The woman usually complains of a copious irritating discharge which is worse after she gets out of bed in the mornings, and which is particularly profuse after a menstrual period. It is profuse enough for her to have to wear a cloth. She often complains of feeling sore and raw between her thighs, and states that she has given up hope of being cured, as she has been treated by so many doctors without relief. Sometimes she complains of the odour of the discharge, but this complaint has not been common in this series.

FIGURE XI.

Labia minora have been separated, showing the flow of pus from the introitus which has poured over the perineum and around the anus.

Clinical Features in Typical Cases.

Introitus.—On separating the *labia minora* there is an escape of thin greenish frothy pus (Figure XI). The inner surfaces of the *labia minora* are sometimes covered with red spots, but these spots are not the bright red spots of an acute infection. This thin greenish pus is seen to be coming out of the vaginal orifice and the *carunculæ myrtiformes* are often reddened. A very typical picture is the condition of the urethral orifice (Figure XII). The

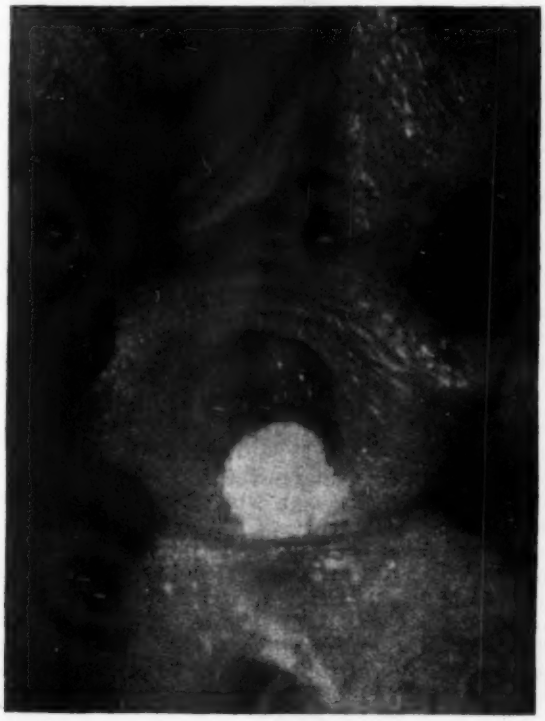

FIGURE XII.

The urethral orifice is reddened, showing that the patient is not cured, and is a typical sign of trichomonas infection. (Piece of wool in vaginal orifice. The urethra is not quite in focus.)

mucous membrane is reddened around the inferior margin of the external meatus, but it is impossible to obtain any urethral discharge on milking the urethra, and the inflammation does not extend into it. This reddening of the urethral orifice is of great importance during the process of cure. I have found that as long as this reddening remains the patient is not cured, and trichomonads can still be found in the vaginal secretion.

Vaginal Walls.—On insertion of a bivalve speculum the vaginal walls are found to be reddened, not the bright red colour of an acute infection, but rather a dull red. The vaginal vaults are often granular and roughened, and are covered with the thin, greenish, frothy discharge, through which can be seen small red spots of inflamed vaginal epithelium.

Cervix.—The cervix is not always affected. Sometimes there is an erosion, but the trichomonas is not the cause of the erosion. Occasionally the cervix is covered with petechia-like spots, the so-called

"strawberry cervix", but there can be a very marked infection by trichomonads with no demonstrable change in the vaginal portion of the cervix.

Vaginal Discharge.—I have mentioned the greenish frothy discharge, which is typical of the condition, and, once seen, it can always be recognized. I have been unable to confirm the presence of the nasty smell, which has been mentioned in the literature.

I have had several patients in whom the discharge was not very profuse and not typical. Clinically these cases were not considered to be due to *Trichomonas vaginalis* infection, as the discharge was whitish in character. However, during the routine examination of the moist film very numerous trichomonads were found; in fact, there were far more parasites per low power field than in the typical infections. Another point in these cases was that the trichomonads varied considerably in size. I am at a loss to explain the reason for this white discharge, but the necessity of examining a moist film in every case of discharge is even more exemplified, more especially if the observer is not familiar with the picture of a stained trichomonad.

Clinical Features in Atypical Cases.

The amount of discharge varies considerably, and in some cases the woman will complain only of a pruritus, and say that she has no discharge. In these cases there is perhaps only very slight reddening of the introitus and vaginal walls.

In other cases the cause of the discharge and pruritus has been thought to be some other condition which was present, such as a relaxed outlet or an eroded cervix. On several occasions I have operated on such patients, and, much to my sorrow, the irritation and discharge have not become less. On further examination I have found, although the clinical picture was not one of typical trichomonas infection, that numerous trichomonads were present. At one period during the last few years I had thought that I was reinfecting some of my patients, as I had several whom I had treated or operated on and whom I afterwards found were infected with trichomonads. I was so worried that I finally thought that my nurse at my rooms had not been thoroughly sterilizing the vaginal specula. However, on investigation, I found that the sterilization was being done properly and that really the infection had been present originally, as well as the other pathological condition.

However, I have now begun to realize that the redness just around the urethral orifice is a typical sign of *Trichomonas vaginalis* infection, and patients with this sign should have the pus or a scraping from the vaginal walls well examined for trichomonads.

Diagnosis.

The method of obtaining the pus is not so important as it was thought to be in the past.

The speculum should not be lubricated, as usually the lubricant contains some antiseptic which will cause the protozoa to die or to lose their motility. This is specially true of the atypical case with very little discharge.

A platinum loop of pus is obtained from the vaginal vault, placed on a warmed glass microscope slide and mixed with several drops of warmed saline solution. A warmed cover slip is applied, and the film is examined under the medium or low power.

In a typical case very numerous trichomonads will be seen and can be picked out by the waving movements of the flagella. Occasionally rewarming the slide will start movements in trichomonads which have become stationary and which have become to look quite like ovoid leucocytes.

In an atypical case it is often difficult to obtain a drop of pus, and in these circumstances I have simply drawn the platinum loop along the vaginal walls and mixed the *débris* obtained with warmed normal saline solution. Usually in this type several fields have to be examined before a trichomonad can be found; but it has been most interesting that after the institution of treatment in these cases the irritation usually disappears, proving, I think, that even a very few trichomonads can be the cause of a very pronounced pruritus. This is the type of case which has been often missed and other causes of pruritus have been sought. This moist drop method can be quickly used, and the true cause for the infection ascertained.

Two other methods of obtaining material for diagnosis can be used. (i) A drop of pus can be taken from off the gloved finger instead of introducing a speculum. I have not used this method, as I consider that inspection of the parts is most important. Even in the very modest patient the insertion of a small bivalve speculum does not cause much inconvenience. (ii) The patient can be asked to send in a specimen of fresh morning urine, and if this is centrifugalized and a drop of the residue is placed on a warm slide, the trichomonads can be seen. This does not mean that the trichomonads are in the bladder, but that a drop of pus from the vagina had fallen into the urine during micturition.

Once the diagnosis has been made, specific treatment must be instituted. Excellent results are obtained usually as far as the discharge and irritation are concerned, but, unfortunately, the symptoms frequently return as soon as treatment is stopped.

Trichomonads can also be seen in stained films, provided the films have been dried in the air and have been stained by the method which has already been described. This method, however, takes much longer than the moist film method. If possible, the vaginal pus should also be inoculated into a suitable medium, as frequently the trichomonads have not been found in the films, whilst after culture numerous parasites have appeared.

Treatment.

The origin of the infection has not been determined; hence the treatment of the condition is at present unsatisfactory. During the last few years numerous

methods of treating the local condition have been used, but nearly all have proved to afford only temporary relief to the condition. As is usually the case, each new treatment gave excellent results in the first few cases. However, each treatment was soon discarded, and a new method was adopted with much the same results.

The first treatment which was used had been advocated by Greenhill,[30] and was very severe; I tried it with but very little success. The external genital organs and vagina were very thoroughly scrubbed with green soap to the point of making them bleed, and then various applications were made to the vagina after it had been dried. I found that very few patients could stand the scrubbing. The applications used by me were boro-glycerine, acriflavine, sodium bicarbonate powder, trypan blue solution, Bonney's blue paint, mercurochrome. I gave this method a very thorough trial, but found that it was practically impossible to repeat the treatment within a week, and that it was a very difficult treatment in unmarried women.

The next treatment tried consisted of washing out the vagina with a saturated solution of bicarbonate of soda and hydrogen peroxide in equal parts. The vagina was dried and a powder of equal parts of quinine sulphate and boracic acid, as advocated by Sure and Bercey,[31] was blown into the vagina with a powder blower. At first the results were very promising, but relapses occurred. I then tried using vaginal suppositories of 5% picric acid as advocated by Goodall.[32] The patient was given eight suppositories and told to insert one each night till she had used the eight, and was then to report again. The vagina was then cleanly swabbed out, and some patients appeared to be much improved. The picric acid was supposed to penetrate the squamous epithelium of the vagina, and masses of desquamated epithelial cells were removed at the "cleaning-up" visit. A 0·5% lactic acid douche was ordered for a week, but relapses very often occurred after the next menstrual period.

I next tried the insertion of tablets of "Yatren 105", as suggested by Rodecurt.[33] The treatment was very easy and the patients inserted one small tablet each night and morning into the vagina. Again some patients were vastly improved. "Yatren" had been used in amœbic dysentery, and it was thought that it would kill the protozoa.

Another method of treatment which I have tried without success was advocated by Rosenthal, Schwartz and Kaldor[34] in 1935. They stated that douching with 25% solution of sodium chloride was most effectual.

Patients appeared to me to be quite cured until the event of another menstrual period, when the infection seemed to light up again and, after the bleeding had stopped, the discharge and irritation were as bad as ever. The trichomonads must therefore have been multiplying very rapidly in the presence of menstrual blood. The usual treatments so far adopted had to be stopped during the menstruation, as the insertion of suppositories or tablets was useless with blood flowing out. I therefore decided that for treatment to be effectual it would have to be applied during a menstrual period as well. Another important factor was that patients themselves were unable to insert the tablets high enough into the vaginal vault.

Present Treatment Used by Author.

The patient is put upon a gynæcological examination chair facing a good light. The introitus is swabbed with a weak solution of biniodide of mercury, and a bivalve speculum is inserted. If a diagnosis has not already been made, a loop of pus is taken and a moist drop preparation is made. The vaginal walls are wiped clean of pus and are swabbed over with a solution of hexylresorcinol, 1 in 1,000.

I use a Graves bivalve speculum and a nasal polypus forceps as a swab-holding forceps. I have found the polypus forceps an excellent instrument to use for this purpose.

The vaginal walls are then dried and four gelatine (size 00) capsules containing a powder of equal parts of "Stovarsol" and sodium bicarbonate are inserted into each vaginal fornix around the cervix. This powder was suggested by Gellhorn.[35] A similar powder is then blown by means of a powder blower all over the cervix, and the speculum is slowly withdrawn, the vaginal walls being kept well stretched whilst the powder is blown into every crevice. The introitus is also covered with powder, especially around the urethral orifice and over the perineum. This technique is repeated every day for a week, and then every other day for another week, and then the patient is told to report after the next period.

Unfortunately, after the menstrual period the patient very often complains that the discharge has returned as badly as ever. If this is the case, she is treated again every other day for another week and given capsules to insert herself at night and morning, and told to report the day her next period commences. The vagina is then swabbed free of blood and capsules are inserted daily whilst her period is present. After the period has finished, she is given capsules which she inserts daily until her next period, when she stops, and reports to me again after this period is over. In the large majority of cases there is no return of discharge or irritation. If irritation occurs, she is told to insert a capsule and usually one or two capsules will give relief.

In a few cases this "Stovarsol" powder stings the patients, and in these I have used tablets of "Devegan", made by Bayer, instead of "Stovarsol" capsules, but I find that they are not so good as the "Stovarsol", although both contain an arsenical preparation.

Results of Treatment.

It is only during the last year that I have realized that the condition of the urethral orifice is a good

criterion as to whether the condition is going to recur or not. If this redness has not cleared up, then I know that the condition will be much worse again after the next menstruation. In some cases this redness takes a long time to disappear, but when the patient no longer complains of irritation or discharge, I find that this spot is no longer red.

The taking of numerous moist smears and the searching for the odd trichomonad as a criterion of cure are, I consider, rather a waste of time, and I have given up this method. Cultivation of trichomonads from vaginal pus is now being used instead.

In several cases no trichomonads were found in direct films, whilst numerous trichomonads have been grown in cultivation of the vaginal pus.

As the source of infection is unknown, cures must be more or less a matter of luck. However, the relief obtained by women who have had irritation and discharge for years and years is, I think, a tremendous factor, even although a proper cure is not effected. I have now records of over one hundred patients treated by me during the last few years, and have a considerable number more at my out-patient department at the Adelaide Hospital. I have managed to keep in touch with most of my private patients and a large number have not relapsed.

In some cases the curing of the discharge and infection has not relieved the irritation. Investigation of these cases has disclosed the fact that the vulval skin has been altered, owing to lack of absorption of vitamin *A* in women who have an achlorhydria. Further work is being done on this subject, but it has been found that patients to whom dilute hydrochloric acid has been given have rapidly lost their irritation, and that the condition of the vulval skin has improved enormously.

Acknowledgements.

My thanks are due to Mr. S. F. Tee, of the Adelaide Hospital laboratory, for his work on the culture and staining of the trichomonads and for his very able photography.

Summary and Conclusions.

1. *Trichomonas vaginalis vaginitis* is a common cause of *pruritus vulvæ* in Australia.

2. The *Trichomonas vaginalis* found in Australia corresponds morphologically with the type that has been described in other parts of the world.

3. A description of the Australian variety of *Trichomonas vaginalis* in its various stages of life history has been given in detail, and the types have been photographed.

4. Methods of staining and cultivation have been described.

5. The effect of ovarian hormone on the growth of trichomonads has been investigated.

6. The risk of infection is much greater since the parasite has been proved to be most resistant to the effects of drying, and a suggestion has been made as to the ætiology of the vaginitis.

7. Real cure is still very difficult, but a new clinical sign of cure has been described.

8. The presence of an achlorhydria with vitamin *A* deficiency is often found in patients in whom the pruritus still persists after the *Trichomonas vaginalis* infection has been cured.

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ILLUSTRATIONS TO THE ARTICLE BY DR. BRIAN H. SWIFT.

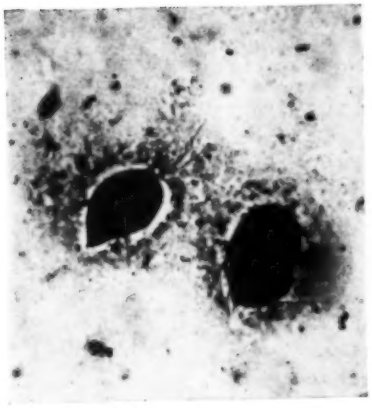

FIGURE I.
Typical small young trichomonads, showing pyriform shape and flagella and homogeneous protoplasm. Gram's stain. ×1,500.

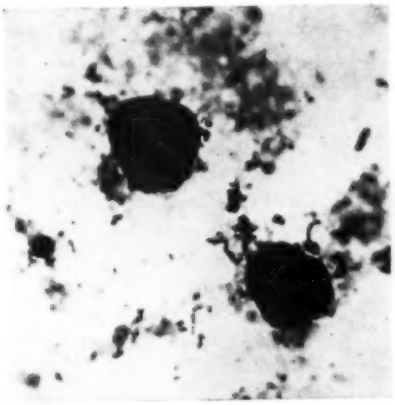

FIGURE II.
Typical young trichomonads. Homogeneous protoplasm. Gram's stain. ×1,500.

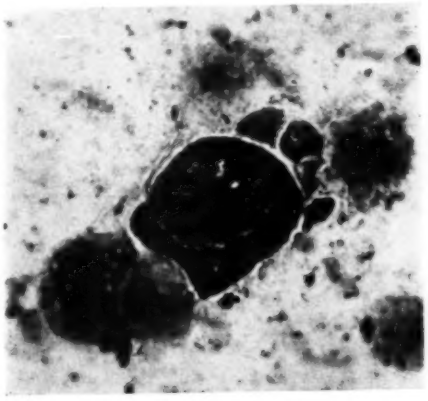

FIGURE III.
Larger, older trichomonad, showing flagella, vacuoles and granular appearance, also more circular shape and having amœboid movement. Gram's stain. ×1,500.

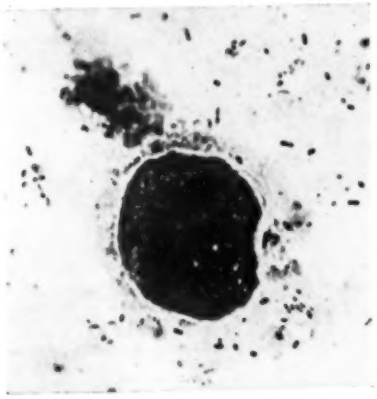

FIGURE IV.
Large old trichomonad, granular with vacuoles. Flagella and nucleus well stained. Gram's stain. ×1,500.

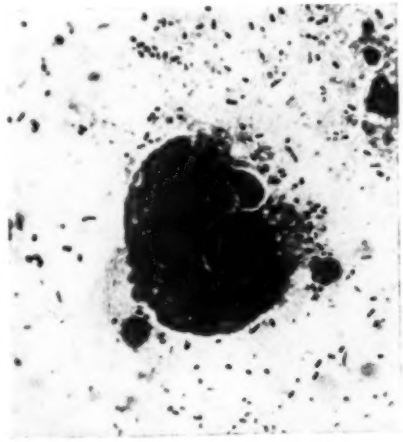

FIGURE V.
Large old trichomonad wrapped around a cell illustrating amœboid movement. Flagella and nucleus can be seen and granular structure of protoplasm. Gram's stain. ×1,500.

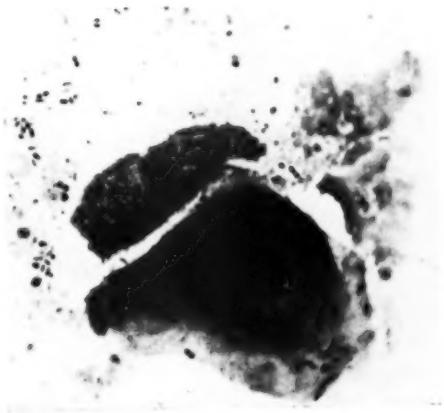

FIGURE VI.
Old large granular trichomonad along side of epithelial cell. Very granular protoplasm. Gram's stain. ×1,500.

ILLUSTRATIONS TO THE ARTICLE BY DR. BRIAN H. SWIFT.

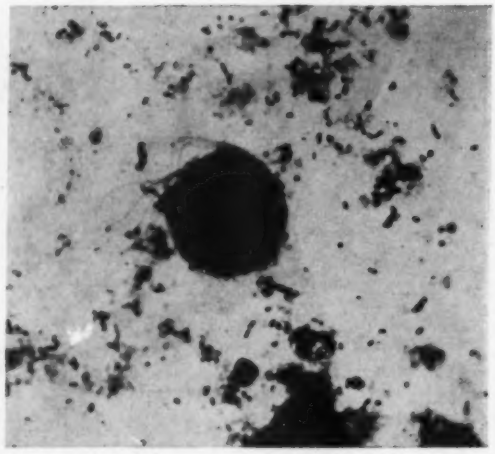

FIGURE VII.

Young trichomonad about to divide by fission, showing two lots of flagella. Protoplasm is homogeneous. Gram's stain. x 1,500.

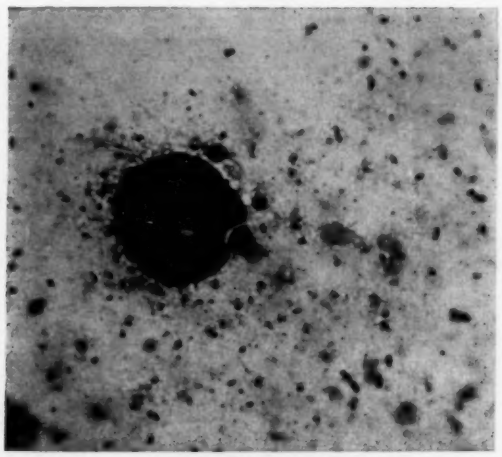

FIGURE VIII.

Middle-aged trichomonad. The undulant membrane and the flagella are well stained. Some vacuolization with granular protoplasm can be seen. Gram's stain. x 1,500.

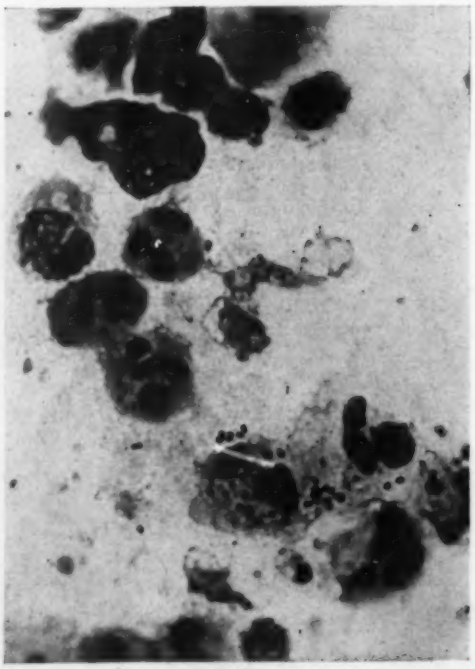

FIGURE IX.

Showing gonococci and a trichomonad (aged with vacuolisation) in the same film. Gram's stain. x 1,500.

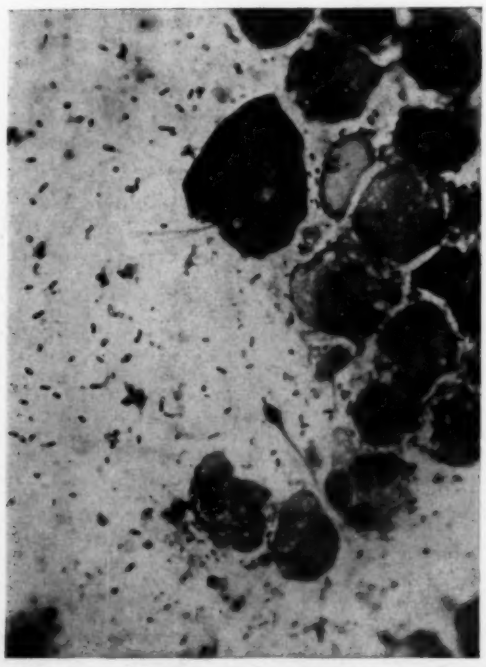

FIGURE X.

Aged trichomonad showing flagella arising from a stoma-like depression. A spermatozoon is also seen. Gram's stain. x 1,500.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. W. S. LAURIE.

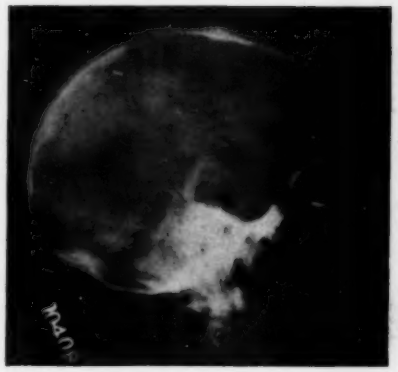

FIGURE IV.
Early skiagram of skull, showing bony thickening, protrusion of lower jaw and pituitary fossa changes.

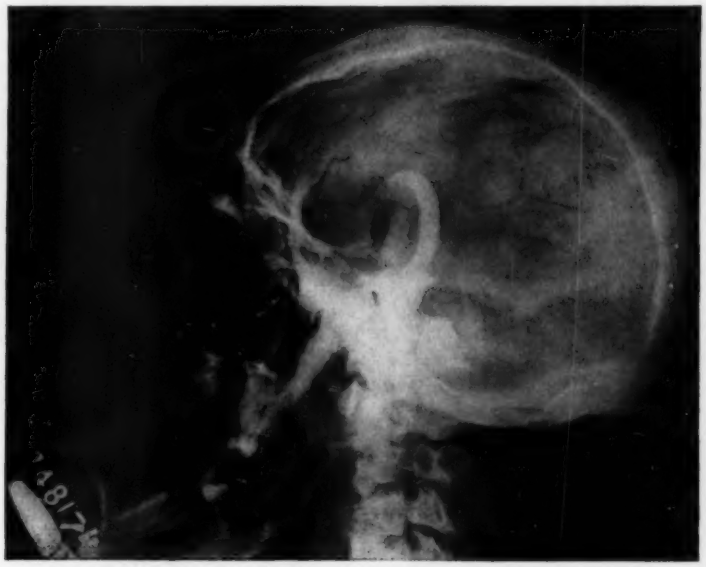

FIGURE IVA.
Skiagram of skull taken at a later stage, showing bony thickening, protrusion of lower jaw and pituitary fossa changes.

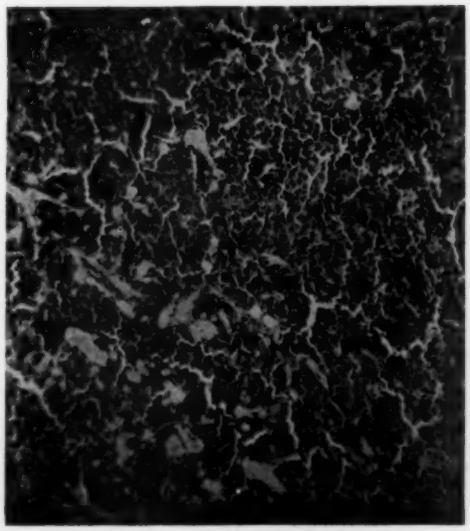

FIGURE VI.
Pituitary—typical eosinophile adenoma, low power view.

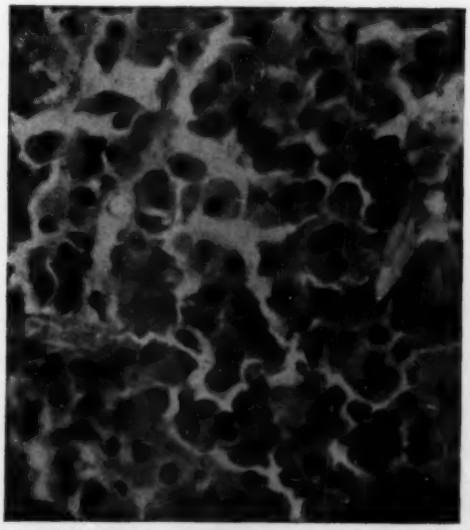

FIGURE VIA.
Pituitary—typical eosinophile adenoma, high power view.

ILLUSTRATION TO THE ARTICLE BY DR. DOROTHY GEPP.

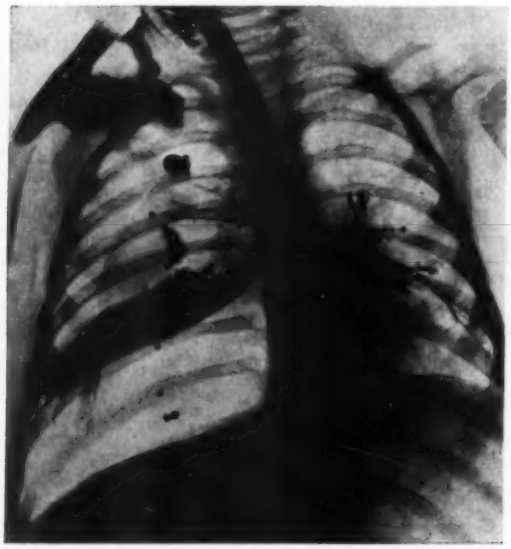

FIGURE II.
Radiogram showing the collapsed right lung after the injection of lipiodol *post mortem*. Globules of lipiodol are passing through the abscess cavities into the space of the pyopneumothorax.

ILLUSTRATION TO THE ARTICLE BY DR. RICHARD FLYNN.

FIGURE I.

ILLUSTRATIONS TO THE ARTICLE BY DR. ALAN H. PENINGTON.

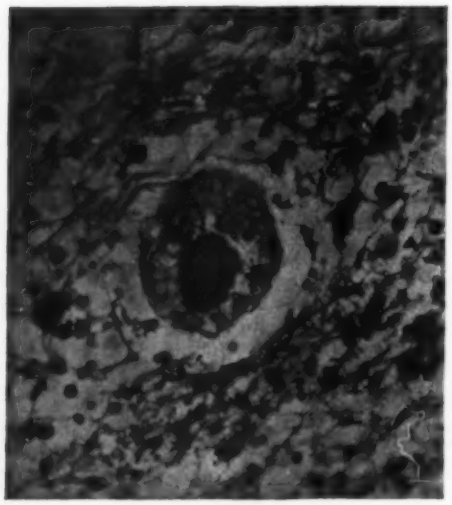

FIGURE II.
Photomicrograph of left frontal cortex (Weigert Pal stain).

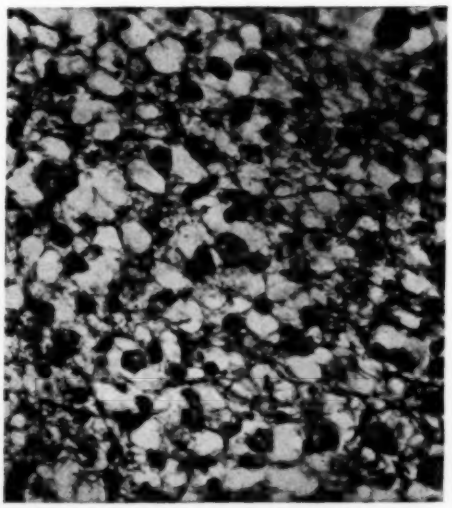

FIGURE III.
Photomicrograph of left internal capsule (Weigert Pal stain).

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Reports of Cases.

A CASE OF ACROMEGALY, WITH CLINICAL AND POST MORTEM FINDINGS.

By H. W. S. LAURIE, M.B., B.S. (Melbourne).
(*From the Baker Medical Research Institute, Alfred Hospital, Melbourne.*)

As the opportunity of correlating the clinical and morbid anatomical findings in acromegaly only occasionally arises, the following report of a well developed case of the progressive type, associated with a typical, progressively enlarging eosinophile adenoma of the anterior pituitary, may be of interest.

History.

The patient, W.D., a male, aged thirty-nine years, first presented himself in 1933 at the out-patient department of the Alfred Hospital. He gave a history of twelve years' progressive enlargement of head, feet and hands. He was at that time sexually potent and had four children, the youngest aged three years. There was no thirst or polyuria, and no undue drowsiness.

Examination showed that the lower jaw was enlarged, the skin of the face and head was thick and pitted, and there were coarse folds on the scalp. The tongue was hypertrophied, the hands and feet were enlarged, and fingers and toes were broadened. The heart, lungs and abdomen showed no abnormality. The urine was clear, the optic disks were normal, and the visual fields were full. Skiagrams of the skull revealed moderate enlargement of the pituitary fossa (Figure IV).

The patient attended hospital at intervals until 1936, during which time there was steady progression of symptoms and signs, so that he was now excessively drowsy and would fall asleep at his work; his sexual function had failed and he was increasingly breathless. The hypertrophy of his tongue was now extreme and obstructed respiration, especially during sleep, when he would awake every few minutes with severe dyspnœa and cyanosis. He also had difficulty in speaking and eating (Figures I, II and III).

Examination now disclosed gross enlargement of the whole head, specially marked in the lower jaw. The heart showed marked general enlargement, and there were occasional extrasystoles. The chest was huge and barrel-shaped, and there were moist sounds at the bases of both lungs. The liver edge was three fingers' breadth below the costal margin.

Skiagrams of the skull now revealed pronounced bony overgrowth throughout, with enlargement and protrusion of the lower jaw. The pituitary fossa was almost completely obliterated (Figure IVa). The optic disks and visual fields, however, remained normal.

Special investigations were carried out as follows. The cells, chlorides, total protein and globulin of the cerebrospinal fluid were normal and the colloidal gold test gave a normal result; the Wassermann test gave no reaction. The blood calcium, phosphorus and cholesterol were normal. Electrocardiograms taken at a late stage showed right-sided preponderance.

In June, 1936, owing to the patient's increasing discomfort, and at his own request, in the hope of preventing progression of the disease, operation was undertaken. Under intratracheally administered gas a right frontal approach was made to the pituitary and the major portion of the gland was removed. Death occurred from myocardial failure four hours after operation.

FIGURE I.
Patient, aged twenty-five years, showing characteristic early facies.

Post Mortem and Histological Findings.

The heart weighed 1,140 grammes (38 ounces); it was grossly enlarged, and marked dilatation of the right auricle was present. The valves and vessels were normal. Microscopically, extreme hypertrophy of individual muscle fibres and extensive interstitial fibrosis were found. The lungs showed congestion only; the right lung weighed 900 grammes (30 ounces), the left 600 grammes (20 ounces).

FIGURE II.
Patient, aged forty years, showing marked progression.

The liver weighed 2,520 grammes (84 ounces) and was considerably enlarged and congested. The gall-bladder was normal. The spleen was large and fibrous and weighed 810 grammes (27 ounces). The left kidney weighed 390 grammes (13 ounces), and the right kidney 270 grammes (9 ounces). The right suprarenal showed *post mortem* liquefaction of the medulla, but appeared

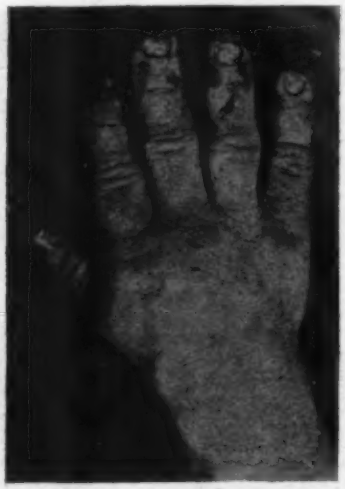

FIGURE III.
Right hand, showing typical broadening.

otherwise normal. The left weighed 14 grammes and showed an abundant medulla and multiple foci of cortical overgrowth. The stomach, intestines, pancreas, bladder and testes showed no abnormality.

The thyreoid gland weighed 210 grammes (seven ounces) and contained multiple adenomata with thick fibrous capsules throughout both lobes (Figure V). In spite of careful examination, no parathyreoid tissue was found.

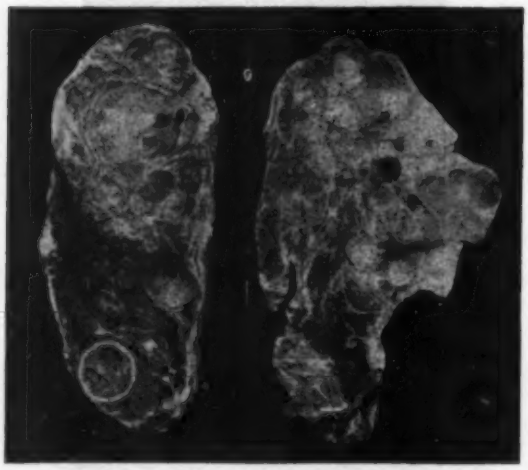

FIGURE V.
Thyreoid showing multiple adenomata with thick capsules.

The brain weighed 1,410 grammes (47 ounces) and presented no abnormality. The pituitary fossa was widened and flattened, and measured 2·5 centimetres (one inch) in diameter; it was the site of recent hypophysectomy, with a small amount of soft pituitary tissue still remaining anteriorly. Microscopic study revealed a typical pituitary adenoma consisting entirely of cells of the eosinophile type (Figure VI).

The skull showed general bony overgrowth, some areas of the vault being 18 millimetres (three-quarters of an inch) in thickness, and extremely sclerotic (Figure VII). Microscopic study showed many bone trabeculæ laid down in the marrow spaces.

Microscopic examination of the liver, lungs, kidneys, pancreas and pineal body revealed no relevant abnormalities except for general congestion.

The causes of death were entered as pituitary adenoma, acromegaly and myocardial failure.

Discussion.

This case presents a typical picture of well-developed acromegaly with steady progression. There are, however, several clinical features worthy of comment. It is of interest that though the pituitary tumour increased progressively in size over a period of years, at no stage did changes in the optic disks or impairment of visual fields appear, in spite of the fact that the tumour extended outside the confines of the *sella turcica*. Cushing and Davidoff[a] reported a case with a large intracranial extension of the tumour without chiasmal involvement.

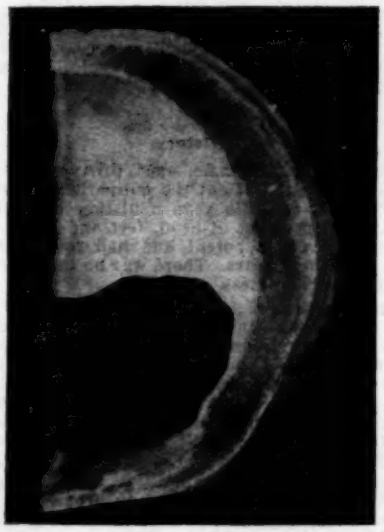

FIGURE VII.
Vault of skull, showing thickening of bone. (The large defect is the result of the recent operation.)

Many acromegalics have a lowered sugar tolerance, probably as a manifestation of hyperpituitarism, or possibly of cerebral or pancreatic origin. This patient, at least while under observation, had no glycosuria. We know that in the later stages of the disease signs of hypopituitarism may be mixed with those of hyperpituitarism, and a high sugar tolerance may be developed.

Most of the reported cases show failure of sexual function, and this case is no exception. In some, however, a preliminary stage of increased activity is described (Cushing and Davidoff); there is no suggestion of such a condition in this case.

Blood calcium estimations were carried out on this patient by Dr. L. B. Cox and Miss I. McPhee,[b] and their results, together with those of several other cases, have recently been published. In the present case they were substantially normal.

One of the patient's main complaints, which finally drove him to seek operation in the hope of relief, was the extreme

enlargement of his tongue, which impeded speech and eating and obstructed respiration (compare Cushing and Davidoff, Case I).

Death in acromegaly, as shown in case reports, may result from the local tumour and its influence on neighbouring cerebral structures, or it may be the result of secondary changes in other organs due to hyperpituitarism. Thus we find such causes of death as arteriosclerosis and myocardial failure, diabetes, bronchopneumonia *et cetera*. The cause in this case was myocardial failure (accelerated by operation) with an extreme degree of chronic passive congestion.

The pathological findings in general are similar to those of previously reported cases. They consist mainly in a widespread tissue hyperplasia with incidence in special sites, and though in detail these sites vary, they may be grouped under three main heads: (*a*) endocrine, (*b*) visceral, (*c*) skeletal. From the historical aspect the skeletal changes received most attention in the earlier reports, and it was from this viewpoint that Marie, in 1887, gave the disease its name.

There are several reported cases in which complete examinations and measurements of the skeleton have been carried out (Knaggs,[2] Cushing and Davidoff[1]). In this case it was not possible to obtain the complete skeleton, but certain features bear out previous findings. The skull shows the typical bony thickening of the vault with protrusion of the mandible and widening of the angle between the body and ramus. The skiagram (Figure IVA) shows the increased size of the frontal sinus, which has been previously reported (Cushing and Davidoff).

Splanchnomegaly is a frequent finding, but attention is focused on it mainly in more recent reports. The increase in size varies considerably in different cases. In this case typical enlargement of the heart, lungs, liver and kidneys was present, as in Cushing and Davidoff's series. The brain, in agreement with most reports, was within normal limits.

Recent reports also have focused attention on the endocrine changes in this disease.

It is now well recognized that the primary cause of the condition is an eosinophile tumour of the anterior pituitary, though there is some discussion as to whether it is a true local adenoma or generalized glandular hyperplasia confined to one type of cell. The size of the tumour has no definite relation to the severity of the disease, and in some of the earlier cases to be studied the pituitary changes were not of sufficient magnitude to become the main focus of attention. It soon came to be recognized, however, that the pituitary lesion was the primary one. Later still, attention was directed to the constant changes in other endocrine glands, notably thyreoid and adrenal. The typical thyreoid enlargement of a colloid adenomatous type is well shown in Figure V. The adrenal change is said to consist in the presence of cortical adenomata with little increase in the medulla. In this case the left suprarenal (which escaped *post mortem* liquefaction) was twice the normal in weight, and presented an abundant medulla as well as cortical hyperplasia.

The testes are stated to be as a rule atrophic in the late stage. Their macroscopic appearance was normal in this case, but unfortunately no histological examination was made.

The thymus, which is reported as being persistent in some cases, was not found.

In one case in Cushing and Davidoff's series parathyreoid enlargement was found, and they state that only one other report of this could be discovered. Careful search in this case failed to reveal any parathyreoid tissue, and we may conclude that there was at any rate no enlargement of these glands.

Summary.

A case of progressive acromegaly associated with an adenoma of the eosinophile cells of the anterior pituitary is presented and its clinical and pathological features are discussed in relation to previously reported cases. The changes in skeletal, visceral and endocrine tissues, as previously reported, are commented on, and their particular variations in this case are noted.

Acknowledgements.

I wish to make grateful acknowledgement to Dr. R. A. Willis and Dr. L. B. Cox for their help in the preparation of this report, and to Dr. L. A. Love for the skiagrams.

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(1) H. Cushing and L. M. Davidoff: "The Pathological Findings in Four Autopsied Cases of Acromegaly, with a Discussion on their Significance", Monograph of the Rockefeller Institute, Number 22, 1927.

(2) R. L. Knaggs: "Acromegaly", *The British Journal of Surgery*, 1935, Volume XXIII, page 69.

(3) L. B. Cox and I. M. McPhee: "Observations upon the Metabolism of Calcium and Phosphorus in Three Cases of Acromegaly, one showing Osteoporosis", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1936, page 390.

A CASE OF SPONTANEOUS PNEUMOTHORAX FOLLOWING LOBAR PNEUMONIA IN A CHILD OF UNDER TWO YEARS.

By DOROTHY GEPP, M.B., B.S.,
Melbourne.

CASES of spontaneous pneumothorax may be grouped into two distinct classes: the type occurring in patients apparently quite healthy previous to the onset of the pneumothorax and in whom no cause for this can be demonstrated, and the type in which a definite preexisting pathological pulmonary condition can be proven.

The first group, admirably described and discussed in a very comprehensive article by Hans Kjaergaard,[1] is ætiologically divided into two types, one due to rupture of scar tissue in the lung, usually apical in site and probably representing a healed tuberculous lesion, the other resulting from rupture of marginal emphysematous bullæ. The name *pneumothorax simplex* is suggested by Kjaergaard for this group of cases, to distinguish it from the second group, in the ætiology of which rupture of an active subpleural tuberculous focus is said to account for 90%, other less common causes being pulmonary suppuration, gangrene, bronchiectasis and traumatic perforation of lung tissue. This type of pneumothorax occurs more commonly in men than in women, and the maximum incidence is between the ages of twenty and forty. It is stated to be very rare in children. A. J. Scott,[2] however, collected from the literature reports of 181 cases of spontaneous pneumothorax occurring in children. In 28 of these the pneumothorax followed a lobar pneumonia, with fatal results in 59%, and in eight the condition supervened on a bronchopneumonia, with a mortality of 75%. Pertussis, measles, diphtheria, tuberculosis and trauma are cited as other predisposing factors.[3] In view of the comparative rarity of the condition in children, the following case is presented as being of interest.

Clinical History.

The patient, a female child, aged one year and eight months, was admitted to the Children's Hospital, Melbourne, with a diagnosis of lobar pneumonia. Her past history indicated that her resistance to infection was poor, as she had suffered from infantile eczema as a baby and since the age of one year had contracted measles, chickenpox and frequent head colds. On admission she had signs of consolidation of the right middle lobe (confirmed by X ray screening) and in addition was suffering from acute tonsillitis and acute right *otitis media*. She was extremely ill for four days, with high temperature and rapid, weak pulse, but the fever then subsided by lysis and her general condition improved, but the chest signs did not completely clear. The general improvement was not maintained for many days; her temperature rose again and after some time signs of further consolidation at the right apex became evident. One month after the onset of the illness there occurred a sudden respiratory collapse, with cyanosis, dyspnœa and rapid, weak pulse. Oxygen and "Coramine" were administered, with improve-

ment of the cyanosis, but the dyspnœa was unrelieved. Examination the morning after the acute collapse revealed the signs of a right-sided pneumothorax. This side of the chest appeared full and relatively immobile, the percussion note was hyperresonant and the breath sounds were distant and had a somewhat amphoric character, especially over the upper half of the chest. Displacement of neighbouring viscera was evident, the apex beat being localized 1·25 centimetres (half an inch) to the left of its former site, and the liver edge being palpable at the level of the umbilicus (this was partly due to toxic enlargement). Two days later the apex beat was still further displaced, almost to the left anterior axillary line, and crepitations were present in the left axilla. Abdominal distension was pronounced. Evidences of general toxæmia and respiratory distress progressively increased, and the signs of pneumothorax persisted until death occurred six weeks after the onset of the illness and thirteen days from the date of the development of the pneumothorax.

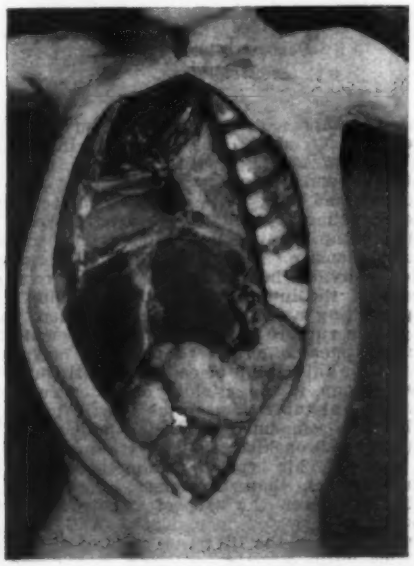


FIGURE I.
Showing the collapsed right lung and empyema cavity.

Post Mortem Examination.

Post mortem X ray screen examination of the chest and injection of lipiodol into the right lung under vision were performed by passing a catheter through a tracheotomy wound down into the right bronchus. The screen showed the upper and lower lobes of the right lung to be collapsed, and the lipiodol was seen to pass slowly into the middle lobe and thence through a perforation into the pneumothorax cavity, dripping slowly down to rest on the diaphragm. The antero-posterior and lateral X ray pictures show very clearly the condition of the right side of the chest just described.

Autopsy confirmed the X ray findings and demonstrated a disintegrated right lung collapsed against the mediastinum, except at the middle lobar region, where it was held to the lateral thoracic wall by a fibrous adhesion, this dividing the pneumothorax cavity into two compartments. In the lower of these compartments were 180 cubic centimetres (six ounces) of thin greenish pus, on which floated a few globules of recently injected lipiodol. The tissue of the upper lobe was soft, purulent, and contained several small abscesses. The heart was displaced to the left. The left lung showed only perihilar hyperæmia and small patches of early purulent consolidation. The *post mortem* appearances are shown in Figures I and II. The sequence of events appeared to be a right lobar pneumonia resulting in the formation of pulmonary abscesses with spontaneous rupture of one or more of the latter, giving rise immediately to a pneumothorax and later to a pyopneumothorax.

Comment.

In its symptoms, signs, course and termination this case typifies the particular ætiological class previously mentioned. The preceding history of a severe type of pneumonia, the sudden onset of respiratory collapse, the presence of widespread tympany, faint amphoric breathing and bell percussion all rendered the diagnosis relatively simple. But in other cases with a more limited pneumothorax, less massive lung collapse and less marked displacement phenomena, the condition must be differentiated from emphysema, from a greatly dilated stomach, and, more rarely, from a diaphragmatic hernia. Emphysema resembles pneumothorax in its tympanitic resonance and feeble respiration, but differs in the mode of onset and in the type of breath sound. X ray examination of the chest and of the stomach following a barium meal will differentiate the other two conditions likely to be confused with pneumothorax.

As occurred in this case, the tendency is towards the secondary development of a pyopneumothorax, and hence the cause is likely to be one of progressively increasing toxæmia, the prognosis grave and the mortality high, as indicated earlier.

The treatment advised is symptomatic, except in those cases in which the pressure of air is sufficient to threaten life, when repeated aspiration is necessary. When fluid or pus accumulates, aspiration or surgical drainage should be performed. However, the general condition of the patient, even after preliminary blood transfusions, is usually so poor as to render doubtful the outcome of any surgical procedure.

Acknowledgements.

I wish to express my thanks to Dr. S. W. Ferguson for permission to report this case, and to Dr. S. W. Williams, who performed the *post mortem* lipiodol injection and the autopsy.

References.

(1) H. Kjaergaard: *Acta Medica Scandinavica*, Supplement Number 42.
(2) A. J. Scott: *Transactions of the Section on Diseases of Children of the American Medical Association*, 1928, page 59.
(3) S. J. McClendon: *Archives of Pediatrics*, August, 1931, page 511.

A CASE OF ACUTE SCHILDER'S DISEASE.

By ALAN H. PENINGTON, M.B., B.S. (Melbourne),
Melbourne.

SCHILDER'S DISEASE is a comparative rarity in Australia, especially in an acute form, and the following case presents some features which are difficult to reconcile with the generally accepted syndrome of the disease. In view of these features, it was considered desirable to report the case for consideration.

A male, S.H., aged forty-eight years, formerly a labourer, was admitted to the pulmonary tuberculosis wards of the Austin Hospital on September 24, 1935. He had been ill for nine weeks previously, complaining of severe cough and loss of weight following on a so-called influenzal attack. These symptoms were associated with night sweats and a fair amount of expectoration. In view of these symptoms, he reported to the Tuberculosis Bureau on September 2, and an X ray examination of his chest revealed the presence of tuberculous infiltration at the apex of the right lung. He was therefore sent to Gresswell Sanatorium, where he was admitted on September 3. Examination at this institution disclosed the absence of the right eye, which had been enucleated as the result of an accident five years previously, and also the presence of paralysis of the

left half of the palate. This palatal paralysis had apparently been unproductive of symptoms, and he appeared to be normal in all other respects, save his pulmonary condition and the loss of his right eye.

When he was admitted to the Austin Hospital on September 24 he stated that three weeks previously, while an inmate of Gresswell Sanatorium, he had noticed a peculiar tingling in his left hand, which he considered at the time to be due to leaning on his left elbow while in bed. This, however, persisted until two days later, when he found himself unable to lift a cup of tea in his left hand. He still considered this to be merely transient, until three days later, when he found that as he ate, food appeared to catch in his left cheek, and that his left leg had "gone to sleep". Within a week he was unable to move his left arm or left leg, and "felt dead" down the left side of his body, and his urinary control had gone, so that he was incontinent of urine, though the anal sphincter was still under voluntary control.

Examination on September 24, at the time of his admission to hospital, revealed a thin emaciated man of cheerful aspect, with a shield covering the right eye, and with a pronounced droop on the left side of his face. He had definite signs of bilateral pulmonary disease, with numerous fine râles at the apices of both lungs and with prolongation of the expiratory murmur. The movements of the left eyeball were normal, and the pupil reacted normally, both to light and accommodation. The right eyeball had been enucleated. The left half of the face was paralysed, but the patient retained the ability to wrinkle the left side of the forehead. The left half of the palate was immovable, and the tongue deviated to the left when protruded. The visual field of the left eye was, to ordinary tests, not limited. The left arm and the left leg were quite immobile, and the muscles in these limbs were spastic, the left leg being extended. The ankle, knee and biceps reflexes were hyperactive on the left side, and knee and ankle clonus were easily elicited. There was a complete loss of sensation over the left side of the body, except for appreciation of deep pain in the limbs, joint and muscle sense as well as superficial sensation all having completely disappeared. Joint and muscle sense on the right side of the body appeared normal, and there was no apparent loss of superficial sensation. The movements of both right arm and right leg were strong, and there was no evidence of increased muscle tension. However, both plantar reflexes were of the Babinski type, and the superficial abdominal reflexes could not be elicited on either side of the body. The patient was incontinent of urine. Other systems of the body were clinically normal.

The day after his admission to hospital, September 25, the patient complained of a tingling sensation in the right foot, as though the foot had "gone to sleep", though examination of his superficial sensation revealed no apparent change. A lumbar puncture was performed on this day, and the cerebro-spinal fluid, as far as could be determined without a manometer, was not under any increased pressure. The fluid itself contained 15 lymphocytes per cubic centimetre, and was clear and colourless, and did not form a pellicle on standing. There was no globulin present in the fluid, and a colloidal gold curve was completely "negative" (000000000). The Wassermann test gave no reaction with either blood or cerebro-spinal fluid. The temperature was not elevated above 37·2° C. (99° F.).

On September 26, two days after his admission to hospital, there was a definite weakness in the flexors of the right foot, associated with loss of appreciation of light touch over the right foot and ankle, the distribution of the anæsthesia being "sock like" in nature. Within four days there was a complete paraplegia in extension, with loss of all superficial sensation to the level of the tenth thoracic nerve; although deep pain could be felt in the right leg, the left leg was completely anæsthetic. At this stage the patient developed a complete retention of urine, and a catheter had to be tied into the urethra.

The condition remained stationary until October 3, when, during the fourth week of his acute illness, the patient developed a complete left temporal hemianopia, but retained normal pupillary reactions to light and accommodation. The paraplegia did not progress any further at this stage. Examination of the *fundus oculi* revealed no abnormality other than a slight cupping of the optic disks, the veins not being engorged. On October 5 the temperature commenced to rise, and the patient became hyperpyrexic. He died on October 7 with a temperature of 40° C. (104° F.), remaining of cheerful aspect until a few minutes before death.

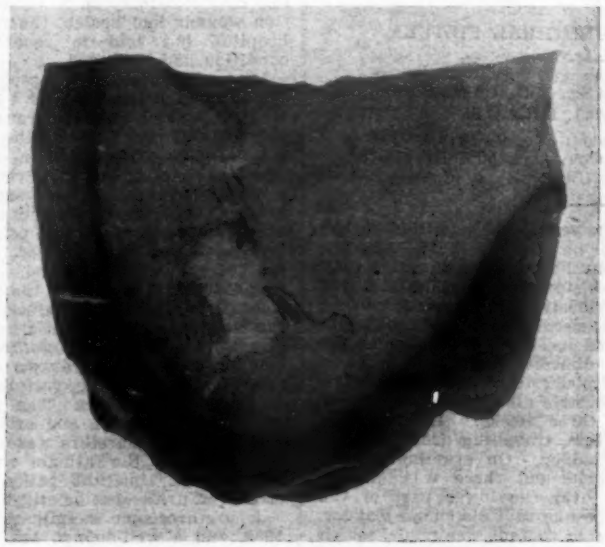


FIGURE I.
Section of brain in left frontal region.

The case history and the progress of symptoms considerably clouded the diagnosis before death. The extreme rapidity of progress and the associated cheerful aspect of the patient pointed in divers directions, while the little assistance derived from lumbar puncture produced many ingenious attempts at explanation. The age of the patient, forty-eight years, was one of the main objections to an *ante mortem* diagnosis of Schilder's disease, though the diffuse nature of the lesions, the absence of any marked temperature disturbance, and the negative findings of the cerebro-spinal fluid all were opposed to a diagnosis of encephalitis. The development of a hemianopia was one of the main features suggestive of this diagnosis prior to death, especially as it was unassociated with any demonstrable change in the optic disk. A suggested parallel between this case and the four cases of *neuromyelitis optica* described by Walsh[1] cannot be maintained in view of the altered time relations, sequence of symptoms, lumbar puncture findings, and the absence of any optic atrophy or papilledema.

It was, however, left in the hands of the pathologist to indicate the exact nature of this progressive degeneration of the cerebral centres. The report on the brain was as follows:

> Specimen of the brain shows on the external surface some swelling of the convolutions, particularly in the right upper frontal region, where one is semi-circular in section and one and a half inches in width, cut at right angles to its direction. This degree of swelling becomes reduced towards the frontal and occipital poles. Over the left hemisphere no such swelling is present. The *pia mater* over the right side is slightly more opaque than normal, while that on the left side is normal.

On section the substance of the brain on the right side shows a very well marked softening, particularly in the frontal and parietal regions. The whole of the internal capsule from optic radiation to frontal radiation is soft and diffluent. On the left side there is some softening, but it is scarcely discernible in the internal capsule.

Microscopically the areas affected show varying grade of demyelination of fibres, which in the right internal capsule and right frontal cortex is complete. In these areas there are present large numbers of phagocytic cells containing fat droplets and around some of the vessels on the right side there is an accumulation of these cells.

The pathological diagnosis is one of *encephalitis periaxialis diffusa* (Schilder's disease).

Reference.

(1) Frank B. Walsh: "Neuromyelitis Optica; Anatomical-Pathological Study of One Case; Clinical Studies of Three additional Cases", *Bulletin of the Johns Hopkins Hospital*, Volume LVI, April, 1935, page 183.

A CASE OF RECTO-URETHRAL FISTULA.

By RICHARD FLYNN, B.A., B.Sc., M.D., Ch.M. (Sydney), M.S. (Minnesota), F.R.A.C.S.,

Honorary Assistant Surgeon, Royal Prince Alfred Hospital; Honorary Surgeon, Lewisham Hospital; Tutor in Surgery, the University of Sydney.

R.G., a male, aged forty-six years, was admitted to the Royal Prince Alfred Hospital on April 26, 1925, suffering from retention of urine, urethral stricture, perineal fistula, and incipient uræmia. His history was that he had had gonorrhœa three years previously. Two years later a perineal abscess had been incised and a urinary fistula had resulted, which had persisted up to the time of his admission. He had noticed difficulty and slight pain on micturition, associated with moderate frequency. He had suffered from dyspepsia, epigastric discomfort and loss of appetite for the preceding three weeks. He had also noticed a pain in the right loin, travelling around to the front and down over the bladder. On examination his tongue was furred, but moist, and there was dulness extending to the umbilicus over the lower part of the abdomen, which was felt to be due to a distended bladder. An estimation of the blood urea content was made, which showed 200 milligrammes of urea per 100 cubic centimetres. A suprapubic cystotomy was done, and when his general condition had improved, dilatation of the stricture was commenced. On May 18, 1925, internal urethrotomy was performed, and at weekly intervals thereafter urethral bougies up to number $^{12}/_{18}$ were passed up to the time of his discharge from hospital on August 10, 1925. He was instructed to return every fourteen days for dilatation, but he was lost sight of till September 18, 1933. On that date he consulted me because he was passing fæces, gas and blood through his penis and urine through his anus. He also stated that he had been very well after his operation until ten months previously. Since that time he had noticed he had to strain hard to pass his urine and that the stream had become narrow; he had also been disturbed with nocturnal frequency of micturition. Examination of his urine showed the presence of blood and fæces. He was readmitted to the Royal Prince Alfred Hospital, where the following investigations were carried out.

A radiograph of his urinary tract showed that no urinary calculus was present. X ray examination after a bismuth enema showed the contour of the sigmoid to be perfectly normal, as was the rest of the large bowel, thus excluding diverticulum of the sigmoid as the cause of the fistula. The red blood cells numbered 4,700,100 per cubic millimetre, the hæmoglobin value was 82%, and the colour index 87; the leucocytes numbered 22,900 per cubic millimetre. The urea content of his blood was estimated at 90 milligrammes per 100 cubic centimetres. His urea concentration figures were low, below 1% of urea at the end of the second hour. His general condition was only moderately good.

Urethral bougies were passed and the stricture was dilated sufficiently to allow the passage of a cystoscope. Cystoscopic examination failed to show the presence of any fistula opening into the bladder; but on withdrawal of the cystoscope the posterior urethra was noted to be greatly inflamed.

A proctoscopic examination of the rectum and anal canal was made, and drops of urine were seen coming through the anterior rectal wall; this was confirmed after an injection of indigo-carmine, when the blue was seen clearly. It was impossible to pass a probe into the rectal end of the fistula.

As the opening of the fistula into the urinary tract had not been seen, although it was felt that it was in the posterior urethra, a urethrogram was made (Figure I). This showed that the fistula opened into the prostatic part of the urethra. On October 19, 1933, a suprapubic cystostomy was done. The lower bowel was put at rest by a left iliac colostomy on November 9, 1933. After the operation wounds had healed, the patient was discharged from hospital, but told to report back when his general condition had improved.

He was readmitted to hospital on January 11, 1934. In the interval he had gained 4·5 kilograms (10 pounds) in weight. A thick ring of fibrous tissue was felt surrounding the urethra, just distal to the peno-scrotal junction.

On January 13, 1934, an attempt was made to pass urethral bougies; but the stricture was impassable even to filiform bougies. Accordingly, on January 18, 1934, external urethrotomy was done, and a large rubber catheter was passed through the penis into the bladder. The perineum was then incised, the prostate was dissected free of the anterior rectal wall, and the fistulous tract was divided. The hole in the rectal wall, which was small, was surrounded with a purse-string suture and invaginated; the hole into the prostatic urethra was closed over with prostatic fascia. The *levatores ani*, together with neighbouring tissues, were drawn across and stitched between the prostate and the rectum, so as to form a barrier between these organs. The penile catheter was changed on January 18, 1934, and every fifth day subsequently, at which times the urethra was also dilated with bougies. On March 3, 1934, the catheter was finally left out altogether, and at that time the patient was able to pass a good stream of urine; but he still had some difficulty.

A urethroscopic examination was made on March 22, 1934, and a well-defined but localized stricture was seen at the peno-scrotal juncture. A week later internal urethrotomy was done, and the stricture was dilated to take a number $^{24}/_{27}$ sound. Dilatations were repeated at weekly intervals. On November 29, 1934, a clamp was placed on the colostomy spur and it fell off on December 10, 1934. The patient's bowels began to act through the natural channel soon afterwards. The colostomy gradually became smaller, and it was closed on September 12, 1935. He was discharged with the wound healed on October 9, 1935. A urethrogram at this stage shows the urethra to be almost of normal calibre with no sign of fistula into the perineum or rectum.

TREATMENT OF PEPTIC ULCER BY EMETINE INJECTIONS.

By J. D. HERLIHY,

Honorary Physician, Lewisham Hospital, New South Wales.

IN view of the intense interest displayed at the present time in the treatment of peptic ulceration by injections, the following preliminary report may be of value.

In July, 1935, owing to the small supplies and also the high cost of "Larostidin" at that time, the writer was

prompted to follow the method described by A. E. Olpp (*vide Medical Record*, November 7, 1934, page 472), in which emetine is used by intravenous injection. The dose given was 0·06 gramme (one grain) of emetine hydrochloride dissolved in six cubic centimetres of distilled water.

One injection was given on alternate days until six were administered. Then after an intermission of seven days a further two or three were given. At the time of injection the stomach must be empty. Should slight dizziness or nausea occur, this can usually be prevented by the prior administration of bicarbonate of soda in a little water. None of my patients had any upset.

All patients were kept on a bland salt-free diet of milk, eggs, cream, cheese, custard, white bread and cream soups whilst receiving the injections. Thereafter they had a free diet with certain restrictions, such as the prohibition of alcohol, roughage and tobacco. Olpp treated 400 patients with 30 recurrences (all the patients with recurrences being alcoholics).

The following are detailed notes on two cases in which a full follow-up was possible; the remaining patients all improved under active treatment, but their present condition is unknown.

Case I.

M.H., aged forty-five years, a male, a relief worker, complained of pain about the epigastrium for four years. The pain was always worse in the small hours of the morning; it was always relieved by food and later by an alkaline powder; he had lost weight.

An X ray examination on July 2, 1935, revealed a constant irregularity of the duodenal bulb, indicative of ulcer, associated with quite a degree of pyloric spasm. The diagnosis was confirmed by a fractional test meal and by a test for occult blood.

Injections were begun with almost immediate relief of symptoms, and despite the fact that the patient was under severe conditions (being on the dole in the first part and relief work conditions to date) the improvement continued, and the radiologist reported on September 11, 1935, that some pyloric spasm still remained; the appearance suggested that the ulcer was not yet healed.

On May 6, 1936, he reported that the duodenal cap was still markedly deformed and there was some increase in the gastric motility. The duodenal cap was not tender and the deformity was probably permanent and due to scarring.

Clinically this patient is free of symptoms and has regained his normal weight, and at this date is in active work in the country.

Case II.

E.M., aged forty-two years, a clerk, complained of loss of weight and of pain in the epigastrium for over twelve years. He had no vomiting; and obtained relief by food and alkalis. His appendix was removed twelve years ago without improvement in his condition. He has been subject to attacks of migraine (average being one attack a week) since he was seven years old.

A radiological report on July 26, 1935, described a persistent gross deformity of the duodenal cap indicative of chronic ulcer. There was no obstruction, and motility did not appear to be increased.

The findings were confirmed by an occult blood test and by a fractional test meal, the figures from the latter being as follows:

Free hydrochloric acid ..	40	24	67	65	64
Total acidity	54	32	80	75	74

The resting juice was 75 cubic centimetres; it contained neither blood nor lactic acid, but gave reactions for bile.

Nine intravenous injections were given, as in Case I, and the same diet routine was followed.

On November 11, 1935, the radiologist reported that a large constant irregularity was still present on the greater curvature side of the duodenal bulb with no localized tenderness. The irregularity was thought probably to be due to a healing ulcer. Evacuation time was normal.

The patient reported next on August 14, 1936, having been quite free of trouble (hence his reason for not reporting); he had put on 9·5 kilograms (one and a half stone) in weight. His migraine ceased with the injections of emetine, and he reported that in the twelve months following there had been only two mild headaches.

Comment.

The cases described are reported mainly in the hope that interest may be stimulated and a further investigation of Olpp's claims may be made. Admittedly the series is far too small to allow any conclusions to be drawn, but it certainly did support his claim not only at the time, but for twelve months after.

The cost, in comparison with that of "Larostidin", is worthy of consideration from the economic viewpoint both in hospital and private practice. The free diet and ambulatory treatment are the same as in treatment by histidine injections.

Case II is of more than passing interest and would appear to suggest that the neurogenic hypothesis is a decided possibility when considering the ætiology of gastric and duodenal ulceration.

Case I is worthy of notice owing to the severe conditions of dole and relief work which had to be contended with.

Reviews.

A MANUAL FOR DIABETICS.

THE booklet "The Principles of Treatment for Diabetic Patients", compiled by Dr. H. Bayldon Ewen, can be perused in half an hour; it contains an elementary description of the main steps in diabetic relief favoured by the author.[1]

The text begins with a rather alarming list of symptoms, which is followed by an outline of the disordered physiology in terms comprehensible by the average patient. As in nearly all examples of this type of manual, however, technical expressions appear which rather defeat the intended ideal of simplicity.

Dr. Ewen apparently still prefers a modified starvation *régime*, with rest in bed for the first week or so of treatment. Short chapters follow on insulin, of which he describes two strengths, 10 and 20 units per cubic centimetre. In this country strengths of 20, 40 and 60 units per cubic centimetre are universally available. The use of the adjective "dangerous", as applied to any form of hypoglycæmic attack, is to be deprecated, as it is this conception which sometimes frightens patients away from the benefits of insulin. Fehling's and Benedict's tests are fully described, but no preference is stated. The final section is called "Practical Points", and should be of the most value, although some of the statements are open to question.

The book should be of definite educational value, but in our opinion requires considerable revision. Small type and the complete absence of illustrations do not add to the interest. Adequate food tables and recipes are a[illegible]ended.

DISEASES OF THE TESTICLE.

"DISEASES OF THE TESTICLE", by Hamilton Bailey, is a small book by a well-known surgical writer.[2] The book, like all this author's other works, is beautifully illustrated by photographs and semi-diagrammatic plates. In general

[1] "The Principles of Treatment for Diabetic Patients", by H. B. Ewen, M.B., Ch.B.; Third Edition, revised and enlarged; 1936. Wellington: A. H. and A. W. Reed. Crown 8vo, pp. 45. Price: 3s. 6d. net.

[2] "Diseases of the Testicle", by H. Bailey, F.R.C.S.; 1936. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 168, with 129 illustrations. Price: 12s. 6d. net.

the work is excellent and is so concise that it contains much more information than most books of twice its size. A pleasing feature is the list of references to standard works at the end of each chapter. This plan always enhances the value of books, and it is a pity that it is not more generally followed. The first six chapters deal with development and the problems associated with imperfect descent of the testes. This is very well done, a full review of the operative procedures being given—the author is a protagonist of operation between the ages of seven and eleven years. In the chapter on method of clinical examination not enough emphasis is laid upon pinching the free *tunica vaginalis* between the fingers, or on the importance of whether or not the epididymis can be felt.

Hydrocele of various types and varicocele are dealt with in a succinct manner, but the chapters contain all that need be known by any except experts.

The chapter on torsion and trauma is excellent and is illustrated by several good case histories. Tuberculous epididymo-orchitis is fully considered and many points of interest are touched upon. It is interesting to note that the author advises epididymectomy instead of orchidectomy in these cases.

"Testicular Neoplasms" is rather a short chapter, but very little of practical importance is omitted. In the differential diagnosis from hæmatocele the importance of the impossibility of palpating the epididymis when there is an effusion into the tunica is not mentioned. The author is in favour of the radical operation in these cases, although he also emphasizes the great value of deep X ray therapy. He advocates the use of the hormone test of the patient's urine for the purpose of estimating the value of this method of treatment.

The book concludes with two interesting chapters, in which the questions of sterility and rejuvenation are shortly considered.

Altogether the book is one of the best small books we have received. There is not a line of extraneous matter in it, and it is packed with clinical information and practical points in respect to diagnosis and treatment. It should be in the possession of every practitioner who meets with lesions of the testis.

ADDENDUM TO THE PHARMACOPŒIA.

On December 29, 1936, the Addendum to the British Pharmacopœia became official.[1] Minor alterations have been made in the monographs, and some have been rewritten, such as those on acriflavine, sterilized water and physiological solution of sodium chloride. The incorporation of new preparations was limited on account of proprietary monopolies or restrictions. The additions include theophylline, but no dose is indicated. Sodium thiosulphate (0·3 to 1·0 gramme) doubtless owes its inclusion to its value in the treatment of toxic manifestations following the use of organic arsenical compounds and salts of the heavy metals. Iodized oil provides an opaque medium for X ray examinations. Aqueous solution of iodine (Lugol's solution) is simply the old *Liquor Iodi* revived with slight modification. Mersalyl is a complex organic salt of mercury; no dose is indicated, but the "injection (10%) of mersalyl" has a dose of 0·5 to 2·0 mils. Mersalyl contains 38·5% to 40·5% of mercury and is one of the mercurial diuretic series. Apparently the theophylline in the injection is to ensure stability. New bismuth preparations are the oxychloride and sodium bismuthyltartrate. The latter has no oral dose, while the former is for administration both by mouth and by intramuscular injection. There is also an injection of the oxychloride containing 0·2 gramme in two mils. Histamine acid phosphate is used subcutaneously to stimulate hydrochloric acid secretion in gastric analysis. A new iron preparation, citrated ferrous chloride (0·2 to 0·3 gramme), is welcome in view of the recent advance in the pharmacology of iron. Two new stramonium preparations, the dry and the liquid extracts, have been added. This is doubtless on account of the extensive use of stramonium in mitigating the Parkinsonian syndrome. The new ergot alkaloid, ergometrine, is included with doses assigned for oral administration and intramuscular and intravenous injection. As ergometrine is only slightly soluble in water, it is presumed that a salt of the alkaloid will be employed, although none is included. Chinioform is used for amœbic dysentery; it is a complex preparation containing not less than 28·2% and not more than 29·6% of iodine. Two new calcium preparations have been added. These are hydrated calcium chloride, appropriate for injection, and calcium gluconate, with only an oral dose assigned. Both preparations should be kept in well-closed containers. Argentoproteinum is of the nature of "Protargol". Two pentavalent organic compounds of arsenic, acetarsol and tryparsamide, also find inclusion. Two new sera are antipneumococcus serum, Type 1 and Type 2; also gas gangrene antitoxin (œdematiens), staphylococcus antitoxin and gas gangrene antitoxin (*Vibrion septique*). Vitamin therapy has received recognition. Concerning *Oleum Morrhuæ*, it is now required that it should contain in one gramme not less than 600 units of vitamin *A* activity and not less than 85 units of antirhachitic activity (vitamin *D*). There is no preparation of vitamin *A* alone. *Liquor Ergosterolis Irradiati* has been deleted and *Liquor Calciferolis* substituted. *Pulvis Vitamini* B_1 is described as an adsorbate of the antineuritic vitamin upon fullers' earth. Ascorbic acid is synonymous with vitamin *C*. Minor matters are the reduction of the maximum dose of cinchophen and an increase in the dosage of iron and ammonium citrate. Both alterations are advantageous.

Altogether the addenum is a very valuable addition to the pharmacopœia.

[1] "Addendum 1936 to the British Pharmacopœia 1932", published under the direction of the General Council of Medical Education and Registration of the United Kingdom; 1936. London: Constable and Company Limited (for the General Medical Council). Demy 8vo, pp. 152. Price: 5s. net.

Notes on Books, Current Journals and New Appliances.

TREATMENT FOR THE GENERAL PRACTITIONER.

EVERY member of the British Medical Association in Australia receives *The British Medical Journal* every week. Among the many valuable contributions published in this journal have been articles on treatment in general practice. Some time ago the first batch of these articles were collected in a single volume; the second volume has now appeared.[1] This volume covers diseases of the nervous system, diseases of the digestive system, diseases of the blood and blood-forming organs, rheumatic diseases, diseases of metabolism and diseases of the kidneys. Medical practitioners will welcome the opportunity of securing under a single cover this series of articles which has been so widely appreciated.

AN AUSTRALIAN MEDICAL DIRECTORY.

THE second annual issue of Knox's "Medical Directory for Australia", for 1936-1937, has been published.[2] Mr. Knox is to be congratulated on its publication. When the first volume appeared it was welcomed by the medical profession of Australia as a valuable book of reference. The second volume is issued on practically the same lines as the first—it is the first volume brought up to date.

[1] "Treatment in General Practice: The Management of Some Major Medical Disorders" (Articles republished from *The British Medical Journal*), Volume II, 1936. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 440, with four plates. Price: 10s. 6d. net.

[2] "Medical Directory for Australia", Second Annual Issue, 1936-1937; 1936. Australia: Errol G. Knox. Demy 8vo, pp. 562. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, JANUARY 23, 1937.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ARTIFICIAL PNEUMOTHORAX.

A FEW months ago pneumothorax therapy in pulmonary tuberculosis was discussed in these pages, the basis of the remarks being an article by J. W. Cutler, published in *The Journal of the American Medical Association*. Cutler maintained that artificial pneumothorax was the most important method of attack that might be employed; the contraindications and dangers were discussed; and the view was expressed that probably adequate use had not been made of artificial pneumothorax therapy by Australian practitioners of medicine. It was further suggested that its advantages should be brought to their notice and that they should be given opportunities for instruction in its technique by post-graduate courses. Further reference to the subject becomes necessary with the publication of a special report written by Dr. F. J. Bentley, embodying the experience of the London County Council in this therapeutic measure.[1] Apart from this report, however, the subject is of such importance, particularly in regard to the selection of suitable cases, that no apology need be offered for bringing it again under discussion. Bentley's report is issued by the Medical Research Council of the Privy Council. As is usual with these reports, the Medical Research Council has published a preface to the report, but it is clearly stated that the views expressed are those of the author and not those of the Research Council or its Tuberculosis Committee. In 1922 artificial pneumothorax therapy was brought prominently to the notice of the medical profession of the English-speaking world by a special report, written by L. S. T. Burrell and Arthur S. MacNalty. A special abstract of this report was published in THE MEDICAL JOURNAL OF AUSTRALIA of November 4, 1922, at page 536. During the fourteen years that have elapsed since the publication of the first report, the production of an artificial pneumothorax has become part of the recognized treatment of pulmonary tuberculosis, and physicians in all countries have adopted it as suitable occasion has arisen. As Bentley points out, however, in his introduction, the wide application of pneumothorax therapy has given rise to a desire for accurate statistical data on the efficiency of the procedure and has also led to the accumulation of material on which information of this kind can be founded. The present report, dealing as it does with 677 new cases that have been followed up for a period of from three to thirteen years, will be studied by all who have extensive dealings with tuberculous patients. In the present instance we propose to indicate the scope of Bentley's investigations and to mention some of his chief findings and general conclusions.

The first part of the report is devoted to a digest of the literature. It must be admitted that Australians have been somewhat backward in contributing to the literature; in the list of one hundred references no article from an Australian journal is cited. It will be useful to reproduce what Bentley describes as the seven deadly sins to be avoided by investigators in this field. He enumerates them as follows:

[1] "Artificial Pneumothorax: Experience of the London County Council", by F. J. Bentley; 1936. London: His Majesty's Stationery Office. Medical Research Council of the Privy Council, Special Report Series, Number 215. Pp. 94. Price: 1s. 6d.

1. *Paucity.*—To draw conclusions from too small a number of cases.

2. *Selection.*—To write up only certain of one's cases.

3. *Precipitancy.*—To report on cases before a sufficient period has elapsed.

4. *False control.*—To evaluate results by comparison with the fate of patients having extensive pleural adhesions.

5. *Equivocation.*—To employ equivocal definitions as to degrees of collapse.

6. *Failure of trace.*—To lose sight of a high proportion of patients.

7. *Lack of detail.*—To fail adequately to describe the extent of disease.

To consider these "deadly sins" will be a salutary exercise for any medical practitioner, for we are all apt, in our enthusiasm to achieve results, to compare cases of almost any disease with one another when we have not sufficient grounds for making a comparison. In pulmonary tuberculosis conditions are extremely difficult, and what Bentley has to say about "false control" is most pertinent. Unfortunately, his remarks cannot be reproduced in full. As an example of what he has to say, we may quote his question as to whether, in general and quite apart from therapy, patients suffering from pulmonary tuberculosis who have a free pleural space have a better prognosis than those whose pleural space is extensively occluded. The answer, he thinks, and we shall all agree with him, must be an undoubted affirmative; and then he adds that, if this is so, we most illogically and unwisely use the latter type of case for purposes of control. Here he quotes Packard, that it is like trying to compare the winners of a race with the losers. Obviously, if a true statistical evaluation of the results of artificial pneumothorax therapy is to be made, a group of patients suited in every way for this type of therapy should be taken; half of them should receive an artificial pneumothorax as part of their treatment, and the other half should be treated in identical fashion, but without the use of a pneumothorax. In addition, the group would have to be so large that chance factors would not need consideration. Human beings cannot, of course, be treated in this way, for if a patient is suited for artificial pneumothorax therapy he must be allowed to have it, and statistical study must go by the board. Even if these considerations are borne in mind, Bentley's study is extremely valuable.

The present study, as already stated, covers 677 new cases followed through a period of from three to thirteen years; of these, 511 have been investigated in considerable clinical detail. The fate of the 677 patients has been compared with that of 3,329 patients treated by conservative methods only. The two groups were classified and followed up in a uniform manner, and were drawn from identical sociological and environmental sections of the population. It is, of course, admitted that this conservative group cannot act as a perfect statistical control. In only 7·2% of the 677 cases had tubercle bacilli never been found in the sputum; and only 259, or 38·3%, of the patients were males. The average duration of pneumothorax therapy in all cases was 18·18 months. All the 677 patients were followed up for a minimum period of three years, and 411 (60·7%) of them were observed for a minimum period of five years. All the 3,329 patients in the conservatively treated group were followed for a period of five years. The results of pneumothorax therapy in the 677 new cases showed a gain in lives of 129·53 at the end of three years; in other words, an increase of 19·1% in the number alive over the expected number amongst those conservatively treated. A sharp distinction is drawn between patients who are undergoing pneumothorax therapy and those in whom the lung has been made to collapse. This distinction is not always borne in mind. Bentley found that incomplete collapse and the presence of pockets dragged down the general level of results. He investigated in detail 267 patients in whom incomplete collapse was produced; only 50% of these survived a period of three years. Of 208 patients in whom collapse was complete, 65·9% were alive after three years; when the disease was strictly unilateral the percentage survival rose to 77·4. It is also important to remember that incomplete collapse may not only fail to produce quiescence of disease, but may, if continued, actually prove to be an added source of danger to the patient.

In what has been written, an outline of some of Bentley's results has been given; on other important aspects discussed by him we have been unable to touch. Obviously great care must be taken in

the selection of patients for pneumothorax therapy; moreover, its inception calls for continuous supervision. The technique of the induction of a pneumothorax is readily acquired. Bentley regards this as a misfortune, for he thinks that the method may be unwisely used; he adds that it is "only on a wide knowledge of the disease in all its aspects that correct selection can be based". The question is open to argument. An inexperienced practitioner will surely send his patient to an expert if he thinks that pneumothorax therapy should be considered. If he undergoes a course of training in this treatment he will realize its limitations and seek advice whenever he is in doubt. Danger might arise if an uninstructed practitioner, fired only by his enthusiasm, were to look on himself as fit to introduce air into the pleural cavity of the tuberculous. Thus arises another point. Bentley states that the London County Council has appointed a panel of five thoracic surgeons, and in addition to equipping theatres at certain of its sanatoria and special hospitals for tuberculosis, has provided thoracic surgical units at two of its general hospitals. These experts will be extremely useful, especially when incomplete collapse only can be obtained. Whether there is need for some such arrangement as this in Australia is a question that may be debated.

Current Comment.

WHOOPING COUGH.

IN the opening sentence of his review of recent work on whooping cough, R. E. Smith states that for children under fifteen years of age the mortality from this disease is at present equal to that of measles, and is slightly larger than the combined rates of diphtheria and scarlet fever.[1] He quotes Greenwood as an authority for this statement. He goes on to state that in some years whooping cough is responsible for two to three thousand deaths in England and Wales and for about six thousand deaths in the United States of America; half of these deaths are those of children under one year of age. Of course, whooping cough also causes a great number of sequelæ that leave their permanent mark on sufferers. It is difficult to form any idea of the incidence of whooping cough in Australia, for it is notifiable only in the Federal Capital Territory and in South Australia. There is no need, however, to quote Australian statistics to draw attention to the seriousness of the condition. Smith's review of recent work in this disease is such a concise and interesting contribution that the attention of medical practitioners may well be drawn to the more important of his statements.

In dealing with any infectious disease it is essential to determine the incubation period. Unanimity has not been reached in regard to this point. Stocks concluded that the incubation period might be as short as three days, but that it was usually a week; Stocks came to his conclusion from an analysis of London figures. Funck-Hellett, after observing seven epidemics in a children's home, believed the incubation period to be longer—ten to twenty days. Smith's findings support those of Stocks, but he wisely adds that the insidious onset makes it difficult to determine precisely when the disease manifests itself. In two boys, reported by H. and E. J. Macdonald, the disease was reproduced experimentally, and the incubation period in both instances was seven days. Infection is usually spread by sufferers who are in the early stages; it is also admitted that carriers exist.

The classical description of whooping cough is that of the worst cases; it must, however, be remembered that the type in which neither whooping nor vomiting occurs, is very common. Smith states that there can be no reasonable doubt that the causative organism of whooping cough is *Hæmophilus pertussis*, more commonly known as the *Bacillus pertussis* of Bordet and Gengou. All bacteriologists will not agree with this statement; many believe that the responsible agent is a virus, and that the *Bacillus pertussis* is a secondary agent. Smith states that the number of those who hold this belief is rapidly diminishing. He points out that the serum of patients convalescent from whooping cough contains complement fixation substances against *Bacillus pertussis*, and that the disease has been reproduced in human volunteers.

Diagnosis is simple when the condition is well established; difficulty sometimes arises in the early stages. The cough-plate method of diagnosis described by Gardner and Leslie is, in Smith's opinion, a reliable and fairly quick confirmatory test. He also uses the findings on examination of the cough-plates to show that the wisdom of the procedure adopted in England in regard to the time during which quarantining of affected children should be carried out. The medical officers of Schools Association advises that patients should be quarantined until the characteristic spasmodic cough and the whooping have ceased for at least two weeks, or, in cases of persistent whooping, for not less than four weeks from the commencement of the spasmodic cough. The Ministry of Health and the Board of Education jointly advised a quarantine period of six weeks from the commencement of the cough. In Denmark, children are allowed to return to school four weeks after the commencement of the spasmodic stage.

From the practical point of view, most interest will probably centre round Smith's views on vaccine and its uses. Widely divergent opinions on the

[1] *The Quarterly Journal of Medicine*, July, 1936.

efficacy of vaccines used for prophylaxis or treatment have been expressed in this and probably every other medical journal that deals with diseases of children; the truth is hard to discover. Smith points out that work on the bacteriology of whooping cough has recently been elaborated so that it is possible to classify the organisms into four phases, all of which are interchangeable by cultural methods, with the possible exception of Phase IV organisms, which are old bacilli, long accustomed to growth on agar. Phase I organisms are those best suited for the preparation of vaccines—these are indeed stated to be necessary for the preparation of useful vaccines. Smith recommends prophylactic vaccination with these organisms in doses of thirty to eighty billions at an early age, preferably between six months and two years of age. He can find no evidence whatever that vaccines are of any value in treatment "except possibly in the earliest stages". He also discusses the use of human convalescent serum.

LATE INFECTION FOLLOWING THE USE OF PINS AND WIRES IN BONES.

THE use of skeletal traction in the treatment of fractures by hooks, pins and wires, has yielded results that were seldom attainable by the old methods. Hooks, pins and wires have become part of the equipment of every surgeon who has to treat fractures, and in certain circumstances their use has become obligatory. At first sight, the insertion of a pin or a wire into a bone seems a simple matter, and doubtless it is. In reality it is the after-treatment that is simplified, for the fractured bone is, as a rule, controlled much more easily than by other methods. The opening of the cancellous tissue of a bone by the insertion of a metal pin or wire is a serious undertaking, and experienced surgeons have learned that cancellous tissue of bone must be treated with respect. For those who have not yet learned this from their own experience, it may be well to draw attention to a communication by S. L. Haas, of the Shriners Hospital for Crippled Children at San Francisco.[1] Haas's paper was read before the Section on Orthopædic Surgery at the annual meeting of the American Medical Association last May. He reported three cases in which latent infections called for operation at periods varying from two to three years after the use of pins and wires. All the patients were children.

Haas points out that though pins and wires are inserted under strict aseptic technique, the tract is potentially infected. Wires and pins have an external opening which in itself acts as a drain and gives some protection. On the other hand, the external opening serves as a portal of entry for bacteria. Fortunately, after the insertion of the wire or pin a defence barrier of granulation tissue is formed along the course of the wire or pin, and helps to inhibit infection. Haas points out that the prevention of motion is important, so that this barrier shall not be injured and new avenues opened up for the invasion of bacteria. For the same reason pins or wires should be removed with strict aseptic precautions and with a minimum of trauma. After removal of the wire, the soft parts heal before the bone, and a sealed-off, firm, non-collapsing channel is left in the bone. Some dormant infection may remain, to light up at a later date. In Haas's cases pathological examination revealed a low-grade infection.

The obvious conclusion is that skeletal traction by pins or wire should not be used if the fracture can be as effectively treated by other means.

[1] *The Journal of the American Medical Association*, November 14, 1936.

THE BIOCHEMICAL LESION IN VITAMIN B_1 DEFICIENCY.

THE title of a recent contribution by the Professor of Biochemistry at Oxford, R. A. Peters, "The Biochemical Lesion in Vitamin B_1 Deficiency",[1] probably forecasts the nature of many medical articles of the future. A new type of analysis is now being undertaken by modern biochemical research, which is comparable in detail and in exactitude in the chemical field to that of present-day histology in the structural. Biochemical investigation into the functional changes of individual cells is possible only when a strictly uniform tissue is available for observation. For this reason, and because of its practical significance, it is natural that most progress has been achieved by study of the cancer cell, especially its glycolysis.

Peters and his staff have striven to reduce to exact biochemical terms the pigeon test for vitamin B_1. It seems that the chance observation of the relation of beri beri to vitamin B_1 deficiency, which was described to us in the physiological classroom, has now been adopted as a standard means of estimating B_1 content of a diet or the potency of antineuritic preparation. Peters points out that a distinct difference is observable between the rates of cure of acute and chronic symptoms in the affected pigeon. Established leg and wing weakness may take many days to improve, whereas opisthotonus and convulsions respond in under an hour following injection of vitamin B_1 under the skull. The story of the search for the biochemical lesion responsible for the acute symptoms is an interesting one. Arrest of all vital and chemical change was produced in the brains of affected pigeons by immersing them in liquid air within ten seconds of bodily death. It was then found that the base of the cerebrum contained a relatively greater amount of lactic acid. Lactic acid in excess of normal had previously been reported in the blood of avitaminous pigeons and beri beri patients. The respiration of normal and avitaminous pigeons' brains was next studied with and without the

[1] *The Lancet*, May 23, 1936.

addition of glucose. It was found that brain respiration was lowered when glucose or lactate was present in the affected bird and raised when the bird had been cured. So far the experiments did not decide absolutely whether the vitamin itself was missing from the tissue, so that the effect of adding pure crystalline vitamin B_1 to the system was essayed, with the result that the oxygen uptake *in vitro* was restored to normal in a quantitative manner. The vitamin, Peters decided, is definitely a catalyst, and the action is so regular that it can be used as a test for the vitamin. Pyruvate is better for this purpose than lactate. Vitamin B_1 appears then *in vivo* to be necessary for the oxidative removal of lactate acid. Two old theories in regard to vitamin B_1 action have been substantiated: (*a*) It affects carbohydrate metabolism at some point related to the three carbon stage, and it is connected with tissue respiration. (*b*) It is a catalyst needed for the oxidative removal of one of the lower degradation products of carbohydrate metabolism. Further work soon gave proof that the vitamin was not a lactate oxidase coenzyme, and that pyruvic acid was an important intermediary metabolite which is normally burned under the influence of the vitamin. Thus pyruvate is found in the blood of the avitaminous organism, either bird or human.

The acute symptoms of B_1 avitaminosis have been definitely related to biochemical changes in the optic lobes and lower part of the brain. Evidence for a toxic cause due to accumulation of lactate or pyruvate is unconvincing, particularly as both are normal metabolites. Thus the purely deficiency hypothesis is strengthened, whereby there is interference with normal intracellular carbohydrate metabolism, with serious detriment to cellular function.

To summarize, therefore, it has been found that in vitamin B_1 deficiency there is a defect in the metabolism of certain carbohydrate intermediates, especially pyruvic acid in the lower part of the brain, which is remedied by minute doses of crystalline vitamin B_1. Both avitaminous pigeon and rat brain, but not normal brain, produce pyruvate *in vitro*. The addition of vitamin B_1 removes this pyruvate. Thus is demonstrated a "biochemical lesion" in the central nervous system which opens up a new vista of laboratory and experimental research, potentially applicable to other insufficiently understood nervous diseases, such as disseminated sclerosis, progressive muscular atrophy, and combined degeneration.

VACCINATION AGAINST PLAGUE.

PROPHYLACTIC inoculation in its most primitive form was carried out with a living virus. For example, in China infants were inoculated with benign smallpox virus; in Turkey slave girls were similarly attended to, so that their skins would be unblemished and they would fetch a higher price. Inoculation with living attenuated cultures has been scientifically employed from time to time in more enlightened years. Several workers, notably Kolle and Otto (1903) and Strong (1906), have experimented with live plague vaccines. Recently L. Otten has conducted an extensive investigation into the prophylaxis of plague with a vaccine of this type.[1] Otten found, as Strong had found many years earlier, that no dead vaccine protected guinea-pigs and wild rats (the most susceptible animals) against plague. He was eventually able to obtain a strain of avirulent bacteria, which he used as a live vaccine with satisfactory results. He determined to inoculate human beings with the same vaccine. He argues that statistics showing the value of the Haffkine vaccine are largely unreliable. Only those people who are willing submit to vaccination; these are on the whole better educated and living under better conditions than those who refuse to be vaccinated; therefore, the two groups are not comparable. Furthermore, during the slow progress of a vaccination campaign many of the unvaccinated become infected, and, at the height of the epidemic, when the mortality is greatest, many of them die. In some epidemics, also, when the disease does not spread diffusely, the high incidence among the unvaccinated inhabitants of a highly infected locality may greatly flatter the figures in favour of vaccination. Otten's method, therefore, was to inoculate every alternate person. In all, 37,000 persons in two large subdistricts in Java were vaccinated during one month. During the following five months, 1·01‰ of the vaccinated and 5·05‰ of the unvaccinated died of bubonic plague. It is possible that the figures would favour vaccination even more if all cases of pneumonic plague could be eliminated with certainty. Otten does not contend that his vaccine can protect against aerogenic infection, "at least when subcutaneously administered at the relatively small dose adhered to till now". It might be mentioned here that no other vaccine confers immunity to pneumonic plague.

As this huge experiment proved beyond doubt the value of prophylactic inoculation with living bacteria, vaccination of the populace as a whole was attempted. During the year 1935 over two million people were vaccinated and 236,000 revaccinated. Some idea of the value of this work may be obtained from a study of the incidence of plague in the Regency of Bandoeng during the epidemic of 1932 to 1935. In each year there was a decrease during the monsoon and an increase during the dry season to a higher incidence than in the previous year, until 1935, when the numbers decreased during the monsoon and remained lower through the rest of the year than at any former period of the epidemic. There are many more features of interest in Otten's valuable paper. It is not proposed to discuss them here. The amazing thing is that such huge numbers of people can be inoculated safely with living organisms. Otten's work may open up a new era in epidemiology.

[1] *The Indian Journal of Medical Research*, July, 1936.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Actinomycosis.

F. H. COTE AND G. R. TINDHOPE (*The Journal of Pathology and Bacteriology*, May, 1936) have encountered four cases of actinomycosis of the ovary during twelve years. In all four cases the right ovary was the first to be involved, and in all there were inflammatory changes in the region of the caecum. Two patients had had operations for appendicitis, suggesting that the alimentary tract was the source of infection in each case. The macroscopic appearances of the three specimens examined were similar. Each had a honeycomb structure, the softened and granulomatous material not having the classical sulphur-yellow granules of actinomycosis, although the ray fungus was present in smears. Each specimen had a definite abscess cavity. One patient recovered from operation, to die later from pulmonary actinomycosis. The other three went down hill from the time of operation, and died within two months.

The Isolation of Organisms of the Typhoid Group.

E. R. JONES (*The Journal of Pathology and Bacteriology*, March, 1936) undertook a series of experiments in an attempt to obtain a more satisfactory method of isolating organisms of the enteric group. It was thought that the combination of a selective fluid medium that would permit the development of small numbers of typhoid bacilli and a selective solid medium that would permit easy recognition of colonies offered most prospect of success. The combination of tetrathionate broth with brilliant green and eosin agar was therefore investigated, and an attempt made to determine the optimum concentrations of brilliant green and eosin for the inhibition of *Bacterium coli* and recovery of *Bacillus typhosus* from (*a*) broth suspensions, (*b*) suspensions in sterilized fæces, and (*c*) unsterilized fæces to which known amounts of a culture of *Bacillus typhosus* had been added. The author finds that *Bacillus typhosus* and organisms of the typhoid group grow freely on lactose-agar with brilliant green and eosin agar at a sufficient concentration of the dyestuffs to inhibit completely the growth of small inocula of *Bacterium coli*. Different specimens of eosin vary considerably in their power to overcome the inhibitory effect of brilliant green, whereas two specimens of brilliant green tried showed very little difference in their power of inhibiting the growth of *Bacterium coli*. Strains of *Bacterium coli* vary in their resistance to the dyestuffs; but all strains examined were more susceptible than *Bacillus typhosus*. As the amount of inoculum is increased, a point is reached at which the concentration of the dye used fails to inhibit *Bacterium coli* completely. *Bacillus typhosus* inoculated into sterilized or unsterilized emulsions of normal fæces can be recovered on the dye plates without loss in numbers. The author states that the combined use of tetrathionate broth and lactose-agar with brilliant green and eosin agar has proved much superior to MacConkey's medium for the isolation of organisms of the typhoid-paratyphoid group from the urine and fæces of patients suffering from enteric fever. Full details of the preparation of the media are given.

Reversed Anaphylaxis in Man.

C. E. KELLETT (*The Journal of Pathology and Bacteriology*, November, 1936) discusses the production of reversed anaphylaxis in man, as exemplified by the development of a series of skin reactions up to six days after the injection of 0·2 cubic centimetre of horse serum into the dermis; the reactions are separated by definite intervals, accompanied by urticaria and erythema, and involve different areas. The author argued that if the antigen were fixed to the cell in graded amounts centrifugally from the site of injection, and provoked a gradually rising titre of antibody, all the observed phenomena could be explained, although he realized that this was opposed to the current belief that the antibody was related to the cell before the anaphylactic phenomena could take place. Experiments were performed with identical amounts of antigen injected into similar areas of skin of the same subjects at intervals of four days. The results tended to show that the combination of circulating antibody and cell-fixed antigen tended to occur in a centrifugal manner. In animals the repeated injection of horse serum at intervals of two or three days may make them refractory to subsequent injection, and Longcope is quoted as stating that by the same method the incubation period for serum sickness may be prolonged for weeks, but that its final appearance may not always be prevented; this agrees well with the hypothesis of reversed anaphylaxis. The author suggests that new avenues of treatment in the so-called allergic states may be opened up if this hypothesis becomes established.

Hypersensitiveness and Antibody Formation in Tuberculous Rabbits.

J. FREUND, ELIZ LAIDLAW AND J. S. MANSFIELD (*Journal of Experimental Medicine*, October, 1936) have studied the relationship between tuberculin skin sensitivity and antibody formation, and their relationship to tuberculous infection. They injected male rabbits with varying doses of the Ravenel strain of tubercle bacilli by either the intravenous or the intratracheal route. Tuberculin tests were carried out every week with a commercial preparation of high potency, 0·2 cubic centimetre of a one in five dilution being used, and the diameters of the erythematous area being read after forty-eight hours. Complement fixation tests were performed every week, a ground suspension of the Ravenel strain of tubercle bacilli being used as antigen. Three groups of rabbits were used, with increasing amounts of infecting dose; all the rabbits died from 102 to 159 days later. The resultant graphs indicate the development of intense sensitization about four weeks after infection, and this was maintained for periods up to three months, after which it was gradually lost before death. No animal reacted to the test within three weeks of death. The antibody graph, on the other hand, showed a fairly rapid development of antibodies from three to seven weeks after infection, and although there was some fluctuation, antibodies were present throughout the whole course of the disease. The intensity of the tuberculin reaction and the titre of the antibody were not parallel, although the animal which showed the most antibody formation also reacted most intensely; the antibodies, however, persisted until death, while the skin reaction disappeared some time before.

Erythrocytes in Acholuric Jaundice.

J. C. HAWKSLEY (*The Journal of Pathology and Bacteriology*, November, 1936) has studied the significance of changes in diameter of the erythrocyte in familial acholuric jaundice. Eight patients were followed, one from birth; the effect of transfusion of normal blood was studied, and four patients were subjected to splenectomy. The infant's mean cell diameter was 7·2μ, and it was symptom free at birth; the red cells, however, showed increased fragility. At five weeks of age pallor and icterus developed, the cell diameter dropped to 6·67μ and by eighteen months of age was varying about 6μ. Before splenectomy in the older patients there was a marked reduction in mean diameter, and the average could be raised by transfusion of normal cells. After splenectomy, in all four cases there was an increase in the mean diameter for about five months; then there occurred a slow fall to a figure midway between the pre-splenectomy figure and the highest figure recorded. The ultimate mean was of normal variability and microcytic in type. The author believes that the conception of familial acholuric jaundice as a disease due primarily to abnormal erythropoiesis is not tenable, nor can the condition be attributed wholly to exaggerated hæmolytic action of the spleen. The presence of fragile cells at birth, before symptoms have appeared, suggests abnormal erythropoiesis as an early factor, and as the swing back to small cells after splenectomy is not accompanied by a positive Van den

Bergh reaction in the blood serum, the development of splenic over-action is a morbid influence that is partially curable.

Tubercle Bacilli in Pork.

W. H. FELDMAN (*Journal of Infectious Diseases*, July-August, 1936) noted that 11% of hogs killed under supervision of Federal meat inspectors showed lesions of tuberculosis; of these, 0·73% showing no generalized lesions, but with small caseous abscesses in hyperplastic lymph nodes, were passed for sterilization. Material from 100 such carcasses was obtained for study, the left precrural gland and the right internal iliac glands being chosen. The glands were immersed in hot water, trimmed and emulsified; four cultures were made into egg yolk medium and four into 3% glycerine; a portion was retained for histological study. Virulent tubercle bacilli were obtained from four of these nodes, and all were of the avian type. Rabbits and chickens were inoculated intravenously, and all developed lesions of the Yersin type. Histological sections from two of the glands from which bacilli had been recovered, showed no morphological evidence of the presence of the bacilli, this being in conflict with the finding of Joest, Noack and Liebrecht that when there is no histological picture of tuberculosis, tubercle bacilli are not present. The author emphasizes the problem of avian tuberculosis, and the fact that virulent bacilli may be present in the carcasses of hogs intended for food, even though all visible lesions of tuberculosis may have been removed.

HYGIENE.

Staphylococcus Toxin, Toxoid, and Antitoxin.

C. E. DOLMAN AND J. S. KITCHING (*Canadian Public Health Journal*, November, 1936) state that the staphylococcus toxin is readily produced *in vitro* and assayed on its hæmolysin content. Most strains are definitely toxigenic, even strains from healthy mucous membranes. This toxin, detoxicated by formaldehyde, has everywhere proved an efficient and harmless antigen, and good results have been claimed against localized staphylococcal infections, such as furunculosis, sinusitis, sycosis, carbuncle, blepharitis, and acne osteomyelitis; the action is rigidly specific. In treatment, a series of doses at five to seven day intervals is given, the dose beginning with 0·5 cubic centimetre or less, and increasing steadily up to one cubic centimetre given subcutaneously. In resistant infections this may be repeated. The treatment is safe, rarely causes troublesome reaction, and should be persevered with in combination with local surgical treatment. Encouraging results are reported with staphylococcus antitoxin strongly supported by laboratory animal tests. Antitoxin is useful against acute staphylococcal infections of skin and subcutaneous tissues, and even in severe generalized infections. Though the action is antitoxic and not antibactericidal, it tends to neutralize the action of the toxin on the defensive leucocytes. Intramuscular doses of fifteen to thirty cubic centimetres are recommended early in definite staphylococcus septicæmia, after preliminary desensitization with smaller doses. Intravenous injection given very slowly in glucose and saline solution drip is suggested in severe cases when the patient is not suffering from bronchopneumonia or a damaged heart or hyperpyrexia. The intrathecal route is chosen in staphylococcal meningitis. Daily repetition of the dose is given if necessary. Rapid rise in temperature, chilliness, even dyspnœa, frequently follow after twenty minutes to one hour. Serum reactions five to fourteen days later are relieved by epinephrine given hypodermically. After recovery the administration of toxoid is important to maintain immunity.

Recovery of Influenza Virus Suspended in Air and its Destruction by Ultra-Violet Radiation.

W. F. WELLS AND H. W. BROWN (*American Journal of Hygiene*, September, 1936) have atomized liquid suspensions of influenza virus in a closed tank, leaving an air suspension of droplets. At intervals samples were withdrawn and, after centrifugalization, were injected intranasally into ferrets. The ferrets were closely observed for signs typical of influenza in ferrets. Blood was taken from the ferrets before inoculation and thirty days later. Virus neutralization tests in susceptible mice were carried out with these sera. The sera of ferrets showing no clinical evidence of influenza did not neutralize the virus. A known potent virus was inoculated thirty days later into the ferrets. Ferrets considered as having had typical influenza remained well; the others developed typical influenza. All ferrets inoculated with virus collected within an hour after suspension contracted influenza (the time interval used always exceeding thirty minutes). Inoculation of samples collected after an hour were unsuccessful. A bacterial testing chamber containing an ultra-violet lamp was built into the sampling line of one of the centrifuges. Whereas the untreated air infected ferrets, irradiated samples withdrawn after ten minutes failed. The viricidal effect of ultra-violet radiation was quite definite.

The Temperature Coefficient of the Production of Erythema by Ultra-Violet Radiation.

JANET H. CLARK (*American Journal of Hygiene*, September, 1936) reports that in human beings equal radiation on the under surface of both forearms was given. During radiation each arm was kept in a water bath—one at 22° C., the other at 32°; the arms were then removed and left at ordinary skin temperature. Erythema appeared at a similar interval of time (slightly earlier in the former). When, however, the two arms after radiation were kept in water baths at 40° C. and 30° C. respectively, the ratio of the velocity of the latent period at 40° C. divided by the velocity of the latent period at 30° C. equals 2·3, the temperature coefficient. Between 20° C. and 30° C. the figure is 1·9. The test of the ultra-violet coagulation of egg albumen gave a temperature coefficient of 8 to 10, the rate being affected somewhat by the pH and by the presence of salts. Erythema is therefore not related to the coagulation of tissue proteins by ultra-violet radiation, but is probably due to a vaso-dilator substance liberated from the damaged tissue. The coagulation of proteins takes place in three stages. First, light denaturation of the protein molecule; temperature coefficient equals 1. The second stage is a reaction with water; temperature coefficient of 10^{+}. The third stage is one of flocculation to form a coagulum.

Control of the Efficiency of Pasteurization of Milk: The Phosphatase Test.

H. D. KAY, of the National Institute for Research in Dairying, Reading, England (*Canadian Public Health Journal*, November, 1936), states that unless milk is retained at a temperature of not less than 145° F. and not more than 150° F. for at least thirty minutes, and immediately cooled to not more than 55° F., it cannot, according to the English milk orders, be properly designated as pasteurized. An exposure of twenty minutes at 140° F. is for naturally infected tuberculous milk dangerously low. Damage to the "cream-line", a commercial objection, results from too high a temperature or too long an exposure. Milk as a biological fluid contains enzymes variously affected by heat. All raw milk contains phosphatase, an enzyme which hydrolyses phosphoric esters, for example, glycerophosphate. It is readily tested and in the correct "holder" process of pasteurization completely disappears. The test is so delicate as to recognize the addition of 0·25% raw milk or a drop of temperature to 143·5° F. Further, phosphatase is somewhat less readily destroyed than the *Bacillus tuberculosis* itself, the most heat-resistant of the pathogenic flora. The extent of inefficiency in processed milk can also be graded in Lovibond blue units. The test can be used for high temperature-short time pasteurization, and is effective in testing both cream and butter. It is rapidly performed and gives clear-cut results. The full details are given in the *Journal of Dairy Research*, November 6, 1935, at page 191.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Women's Hospital, Crown Street, Sydney, on July 23, 1936. The meeting took the form of a series of demonstrations by members of the honorary staff.

Twin Pregnancy Complicated by Bilateral Hydronephrosis and Hæmaturia.

DR. A. J. GIBSON showed a patient who had suffered from twin pregnancy complicated by bilateral hydronephrosis and hæmaturia. This case will be published separately in a subsequent issue.

Pregnancy Complicated by a Ureteric Calculus.

Dr. Gibson also showed a woman whose pregnancy had been complicated by a ureteric calculus. The patient, a woman of twenty-six years, had first consulted Dr. Gibson on December 13, 1935, being then in the eighth week of her first pregnancy; she complained of severe pain in the right iliac fossa. Two weeks before this, attacks of vomiting had occurred, as well as albuminuria, which had cleared up with rest in bed. Six days before Dr. Gibson saw the patient she had been seized with severe pain in the right iliac fossa, morphine being required to relieve it. Subsequently the pain moved round more to the back; the patient's temperature rose to 37·8° C. (100° F.), she had frequency of micturition with slight scalding, and she was very constipated. Examination revealed tenderness, swelling and rigidity over the right side of the renal area. The uterus was enlarged, the pelvis showed no abnormality, but the urine was acid and contained pus and albumin. A catheter specimen was found to contain albumin, a few red cells and a few pus cells; and culture yielded a few colonies of coliform bacilli. A provisional diagnosis of pyelitis due to calculus was made. X ray examination revealed a small opaque calculus in the region of the right uretero-vesical junction. Intravenous pyelography showed both kidneys to be functioning very satisfactorily: a good shadow of the calyces was obtained within five minutes and dye was already in the bladder. After thirty-five minutes there was no evidence of hydronephrosis or other abnormality of the calyces, although the whole of the right ureter was outlined, demonstrating a very mild degree of stasis. The opaque shadow previously noted was believed to be within the right ureter at the uretero-vesical junction and to be the cause of the mild degree of stasis.

Appendicectomy had been performed on this patient five years previously. She had had quinsy when twelve years of age, and every year for some years. Her tonsils had been removed in April, 1935.

With rest, alkalis and foments the pain subsided; eserine was also given; but as some tenderness persisted, Dr. Bridge, on December 24, 1935, passed two ureteral dilators in an attempt to facilitate the passage of the calculus. On December 30, 1935, the urine was free of albumin and no pus was evident macroscopically. As the patient felt well, she was allowed to go home. Since then the pregnancy had been uneventful, and labour was due at any time. An X ray picture, taken on January 7, 1936, showed the calculus to be still present; there was no suggestion of its having passed. Dr. Gibson said that after the delivery another X ray picture would be taken to see if the calculus had passed.

The Toxæmias of Pregnancy.

Dr. Gibson then discussed the toxæmias of pregnancy. He said that Dr. E. S. Morris and Dr. Sandford Morgan, from an investigation of 1,073 maternal deaths occurring from 1929 to 1933, had come to the conclusion that the outstanding problem of New South Wales, and probably of Australia generally, was how to effect a reduction of deaths from the toxæmias of pregnancy. Of the deaths, 25% could be attributed to these conditions, but actually the percentage was much higher, for many cases of puerperal sepsis, *ante partum* hæmorrhage and embolism were complicated by toxæmia. Dr. Morris and Dr. Morgan affirmed that adequate antenatal care and treatment, important as they were, had many limitations in the prevention of eclampsia and toxæmia. They considered that the admission to hospital of a greater number of toxæmic patients appeared to be necessary, and that the treatment given to the patients who died, in the series under review, though suitable, was in most instances administered too late. Dr. Gibson wondered what could be done to reduce the mortality rate in these conditions, the chief of which were pre-eclamptic toxæmia, eclampsia, nephritic toxæmia and essential hypertension.

Dr. Gibson said that the first important point was to find out which patients were liable to develop toxæmia. Any patient who gave a history of having suffered from more than one of the following conditions should be regarded as prone to develop toxæmia at some time during pregnancy: scarlet fever, diphtheria, rheumatic fever, pneumonia, severe influenza, recurring tonsillitis, septic tonsils, quinsy, oral sepsis, chronic infection of the antrum, mastoid, nasal sinuses *et cetera*. Dr. Gibson said that eclampsia might occur at any age, but was most common in young *primigravidæ*; if it appeared before the thirty-sixth week it was more likely to be of a severe type, and the prognosis was less favourable than in eclampsia at term.

The first sign to appear was usually a rise of the blood pressure above 130 millimetres of mercury, systolic, and above 80, diastolic. This rise might precede the appearance of albuminuria or œdema by some weeks. Therefore, the taking of the blood pressure from the first visit onwards was essential. A rise in the blood pressure might occur as early as the second or third month of pregnancy, followed by a variable interval during which the pressure was normal, and then by a permanent rise associated with other signs of toxæmia. In pre-eclamptic toxæmia this early rise was important as an indication that trouble was to be expected later. In nephritic toxæmia the onset of hypertension and albuminuria was earlier than in pre-eclamptic toxæmia, usually before the fifth month. In essential hypertension there was a pre-pregnancy hypertension associated with a generalized arterial spasm of unknown origin. In the early stages the kidneys might not be affected even with a systolic blood pressure of 200 millimetres of mercury, but later the kidneys became sclerotic and chronic interstitial nephritis supervened. The condition was usually aggravated by the pregnancy. In this condition there was risk of the fœtus dying from placental infarction, and concealed accidental hæmorrhage might occur. At the first indication of a rise in the blood pressure the patient should be advised to rest and to restrict her diet, and she should be examined more frequently for œdema and albuminuria. Œdema usually started in the feet and ankles, and should be attributed to toxæmia unless proved due to other causes. The faintest haze of albumin in a catheter specimen should be regarded as ominious.

In regard to prognosis, Dr. Gibson said that patients with pre-eclamptic toxæmia did not usually recover completely before delivery. In the more severe forms about 10% might develop eclampsia, but the greatest danger was that chronic nephritis or chronic hypertension might supervene. Statistics showed that about 10% of pre-eclamptic patients and 5% of eclamptic patients had signs of chronic nephritis or hypertension after delivery, but that considerably more, namely, 50% and 30% respectively, developed toxæmia in each subsequent pregnancy. The incidence of chronic nephritis and hypertension after eclampsia was lower than after pre-eclamptic toxæmia. This was probably because eclampsia was an acute disease usually followed by speedy termination of the pregnancy. Pre-eclamptic toxæmia might go on for weeks. It was found that 75% of these patients developed nephritis or hypertension if pre-eclamptic toxæmia lasted longer than four weeks. Therefore pre-eclamptic toxæmia should not be allowed to continue for more than three weeks from

the time the disease became established. Speaking generally, the older the patient, the greater the amount of albumin, and the higher the blood pressure; the longer the toxæmia had been present before the pregnancy was terminated, the more likely the patient was to suffer from chronic nephritis or chronic hypertension. Favourable signs were the disappearance of albumin and the return to normal of the blood pressure within three weeks of delivery; but final judgement could not be given till at least six months after delivery.

The immediate and remote prognosis in chronic nephritis was bad. The disease process was hastened; expectation of life was shortened. The patient often died of uræmic coma, uræmic convulsions and concealed accidental hæmorrhage. The fœtus often died *in utero* from placental infarction. The higher the blood pressure and the greater the albuminuria, the worse the prognosis. If pregnancy was carried on to try to save the child, the patient must be kept in bed entirely to control the hypertension, and Cæsarean section might be necessary at the thirty-sixth week, with sterilization.

In conclusion, Dr. Gibson said that Stander considered that estimation of the blood uric acid gave the most reliable prognostic indications. Normally the blood uric acid was 3·5 milligrammes *per centum*; if it rose to 5% or over the condition was serious, and if it rose to over 6% eclampsia was imminent. If the blood uric acid was rising, the pregnancy should be terminated, even if clinically the patient's condition seemed to be improving.

Progressive Hypertension with Succeeding Pregnancies.

DR. T. DIXON HUGHES showed a patient illustrating the effect of succeeding pregnancies on essential hypertension. The patient, aged thirty-seven years, had at her first pregnancy a systolic blood pressure of 140 millimetres of mercury, which rose to 200 at seven and a half months, when albumin appeared. Pregnancy was terminated at this stage, and a 2·5 kilogram (five and a half pound) living child was obtained. The systolic blood pressure gradually dropped until it reached 160 millimetres of mercury, that is, 20 millimetres above its original figure. The patient again became pregnant, and the systolic blood pressure rose to 210; before this stage was reached an investigation of her renal function and eye grounds was undertaken, and all were found to be normal, the urea test giving a very satisfactory result. Again the pregnancy had to be terminated at eight months, as the blood pressure continued to rise and death of the fœtus was feared. After confinement the systolic blood pressure fell to 180 millimetres of mercury, that is, 20 millimetres above that of the patient's pre-pregnant condition and 40 millimetres above her original blood pressure reading before her first pregnancy. In spite of advice, she again became pregnant, and after two and half months her systolic blood pressure had risen to 210 millimetres of mercury. Abortion was induced at this stage, the blood pressure falling to 200, at which figure it remained. This case demonstrated that successive pregnancies were harmful to patients with essential hypertension.

The Advantages of Trial Labour.

Dr. Dixon Hughes's next case illustrated the advantages of trial labour. The patient, aged twenty-eight years, had had labour induced four times owing to disproportion—at seven and a half months, eight months, seven and a half months, and again at eight months—the average length of labour being twenty hours. The weight of the babies averaged about 2·3 kilograms (five pounds). The first baby died a few hours after birth and one other child died at a later date. The patient was of small stature, but her pelvic measurements appeared to be normal. Gross disproportion appeared at eight months; this, however, was associated with an occipito-posterior position. A pelvic examination gave the impression that the pelvis was roomy, and this, together with the fact that the patient had never had a trial labour, suggested that a trial labour should be given in spite of the fact that there appeared to be an enormous amount of disproportion. The labour was normal and a 4·77 kilogram (ten and a half pound) baby was born in twelve hours without any artificial aid. This case was one of the many that could be shown to demonstrate that no *primipara* should be subjected to an induction of labour for disproportion without a trial of labour first. Dr. Ridler, commenting on this case, said that the man was not yet born who could decide when the head would go through at eight months and not go through at full time.

Hormone Therapy.

DR. JOHN CHESTERMAN showed a woman suffering from dysmenorrhœa and menorrhagia, whose periods recurred every seventeen days. She had been treated with intramuscular injections of "Proluton", a preparation of the hormone of the *corpus luteum*. The patient's history was briefly as follows: She was thirty years of age, and had been married seven years. No pregnancies had occurred, but contraceptives were used. Her menstrual periods commenced when she was fourteen years old, occurred regularly each twenty-eight days without causing pain, and lasted four or five days. A few years later the menstrual periods caused sufficient pain to compel her to lose time from work. She married at the age of twenty-three years, and after marriage the loss of blood at each period increased. Later she had some bleeding at mid-period time. In 1931 dilatation of the cervix was performed for her dysmenorrhœa, without relief. At this time she had to stay in bed during the first day or two of her menstrual period. In 1933 she developed acute appendicitis, and appendicectomy was performed. In 1934 a section and an operation were performed on her ovaries. The details of the operation could not be ascertained. For a short time she had less pain, but the dysmenorrhœa then increased. When the patient came for treatment, her menstrual periods were recurring about every sixteen or seventeen days and lasting for six days, with heavy loss of blood and severe pain for the first few days of each period. She stated that sterilization by X rays had been advised to prevent the pain and loss of blood. On examination *per vaginam* no gross abnormality of the uterus or appendages was detected, but the ovaries were slightly enlarged and tender. General examination did not reveal any possible cause for the bleeding. It was decided to administer *corpus luteum* hormone on the assumption that the extrusion of the ovum from Graafian follicles was not taking place, and consequently that only atresic *corpora lutea* were being formed. The hormone of the *corpus luteum* being responsible for the "progestational" growth of the uterine endometrium after ovulation at the middle of the menstrual cycle, any deficiency of this hormone would lead to a breaking down of the endometrium and consequent bleeding. Following a menstrual period from February 17 to February 23, 1936, the patient was given one ampoule of "Proluton" by intramuscular injection each day on March 2, 3 and 4. Following these injections she had no bleeding for six weeks, when on April 14 a period lasting six days occurred. It was associated with the usual pain. This long period of amenorrhœa suggested that a smaller quantity of "Proluton" would suffice, so the patient was asked to report early in May for another injection. However, a menstrual period commenced at the end of April; so this injection was not given. She was given one ampoule on May 15 and a second a week later. A menstrual period lasting six days occurred, with only slight pain, three weeks later, on June 12, 1936. Again the patient was given two injections with a week's interval, on June 25 and July 2, and a period occurred on July 18, lasting six days, but again associated with severe pain. It was pointed out by Dr. Chesterman that since treatment had been commenced five months previously, the frequency of the patient's menstrual periods had been reduced to almost normal, and she felt much better in general health. It seemed reasonable to assume that the "Proluton" was responsible for this improvement. Although it was not suggested that this patient's condition was cured, it seemed possible that one injection each month might suffice to control the frequency of her menstruation.

Puerperal Sepsis.

Dr. Chesterman's next patient was a woman suffering from puerperal septicæmia. She was a *primigravida*, aged twenty-five years. Following a normal pregnancy, she was

delivered of a healthy infant early on June 24, 1936, after a labour lasting fifteen hours, at which no pelvic examination was made. There was a small perineal tear which did not require suture. The same day her temperature rose to 37·25° C. (99° F.); on June 26, 1936, it reached 38·35° C. (101° F.), and the following day she had a rigor. Two days later she complained of a sore throat. On July 1 her temperature reached 40° C. (104° F.), and she appeared seriously ill. Similar strains of hæmolytic streptococci were grown from her blood and from her lochia. A blood count on July 1 showed 2,220,000 red blood cells per cubic millimetre, the hæmoglobin value was 42%, and there were 14,200 leucocytes per cubic millimetre. In view of the anæmia, the patient was given a blood transfusion of 400 cubic centimetres of citrated blood. During the three weeks from July 1 to July 21, 1936, this patient was given six blood transfusions. After each transfusion there was apparently a very slight improvement reflected in the temperature and pulse chart, but there was a steady deterioration in the patient's condition. The destructive effect of the hæmolytic streptococci was well demonstrated by successive blood counts before and after the transfusions; the slight gain in red cells following a transfusion was lost during the next forty-eight hours. After the sixth transfusion, by which time the patient had received nearly five and a half pints of blood, the red cell count had risen to only 3,050,000, and leucocytes had dropped to 9,000 per cubic millimetre. At this time the patient was *in extremis*; for ten days her pulse rate had varied between 140 and 160 per minute, with a correspondingly high temperature. On July 21, 1936, she was given 0·6 gramme of "Neosalvarsan" intravenously. Within a few hours her temperature fell to 38·35° C. (101° F.) and the pulse rate to 115, the lowest readings for fourteen days, and she showed slight clinical improvement. At the same time only seven colonies of streptococci grew from one cubic centimetre of blood. Unfortunately, no previous attempt to count the number of organisms in one cubic centimetre of blood had been made. The patient maintained the slight improvement on July 22, 1936, on which day she was given 0·6 gramme of "Myosalvarsan" intramuscularly. Although she was still desperately ill, Dr. Chesterman considered that her chances of recovery had improved during the past forty-eight hours. Should she recover, the repeated blood transfusions had undoubtedly turned the scale in her favour. Dr. Chesterman also said that an attempt to trace the source of this patient's infection had been made by taking swabbings from the throats of the nurses and students who had assisted at her delivery, and a hæmolytic streptococcus had been grown from the throat of a student. The Professor of Bacteriology at the University of Sydney, who had grown cultures from these swabbings, considered the streptococcus to be a strain similar to the infecting organism, and the probable source of infection.

Dr. Chesterman also read the case history and showed the clinical chart of a woman suffering from hæmolytic streptococcal septicæmia following abortion, who had been treated with intravenous injections of olive oil emulsion. The patient, a married woman, thirty-six years of age, had had an incomplete miscarriage when she was eleven weeks pregnant, ten days before her admission to hospital. On admission on December 17, 1935, her temperature was 39·67° C. (103·4° F.), and her pulse rate 140. She was given four doses of quinine sulphate, 0·3 gramme (five grains) at two-hourly intervals, followed by one cubic centimetre of pituitrin injected hypodermically. She expelled several pieces of placenta. Two days later, because of the offensive odour of her lochial discharge, an intrauterine douche was given and a self-retaining catheter introduced into the uterus; the catheter was left in position for twenty-four hours, 57 cubic centimetres (two ounces) of glycerine being injected each six hours. Improvement was manifest during the next forty-eight hours, but the following day (the fifth after admission) the patient's temperature rose to 39·56° C. (103·2° F.). A strain of hæmolytic streptococci was grown by culture from her blood. At 3 p.m. on December 19, 1935, 20 cubic centimetres of olive oil emulsion were injected into the right external jugular vein. This procedure was repeated at 6 p.m. Three further injections of 10 cubic centimetres each were given at three-hourly intervals. About thirty hours later a final injection of 10 cubic centimetres was given. These six injections did not have any apparent effect either on the patient's condition or on the character of her temperature and pulse chart. The patient died on December 26, 1935. A *post mortem* examination revealed the changes usually associated with an acute blood-stream infection. The streptococci had apparently been invading the blood stream from the blood clot over the placental site, since no gross focus of infection was present. This case history was read to stress the point that the injections of olive oil emulsion had in no way influenced the course of the disease towards its fatal termination.

Hypertension.

DR. H. A. MCCREDIE showed a patient whose pregnancy was complicated by essential hypertension. The patient was a *primipara*, aged thirty-six years, who had had an ectopic gestation seven years previously. She had, on rare occasions, suffered from sore throat, and had had all her teeth extracted owing to pyorrhœa. Her last menstrual period was from June 14 to 21, 1935. When admitted to hospital on September 17, 1935, she complained of shortness of breath on exertion. Examination showed her to be fourteen weeks pregnant, with a moderate cardiac hypertrophy, a systolic blood pressure of 200 millimetres of mercury, and a diastolic pressure of 115. The urine showed no albumin, blood cells or casts, and was sterile on attempted culture. The blood urea was 15 milligrammes *per centum*, and the urea concentration up to 3·2%, at a specific gravity of 1021.

A diagnosis of essential hypertension was made, and the patient was discharged from hospital on October 4, 1935, the blood pressure remaining the same. She attended as an out-patient and was readmitted to hospital on November 18, 1935, the systolic blood pressure then being 210 millimetres of mercury, and the diastolic 130. The next day the systolic pressure had fallen to 180 and the diastolic to 110. The urine and urea clearance showed no alteration. X ray examination of the heart and great vessels showed cardiac enlargement involving the left ventricle. The patient was not distressed. The blood pressure remained in the vicinity of 180 millimetres of mercury, systolic, and 119, diastolic, and the patient was discharged from hospital on December 16, 1935.

She was readmitted from the out-patient department on March 23, 1936, being thirty-nine weeks pregnant. The fœtus was in the occipito-anterior position, and the fœtal heart was heard. The systolic blood pressure was 210 and the diastolic 120 millimetres of mercury. There was no albuminuria. The blood urea was 13 milligrammes *per centum*, and the urea concentration was 2·04%, at a specific gravity of 1019. The blood uric acid was 5·4 milligrammes *per centum*. An attempt at medical induction on March 27, 1936, had no effect, and it was repeated on March 29, 1936. A child weighing 3·2 kilograms (seven pounds) was born the following day after a normal labour lasting five hours. The puerperium was normal.

The case was of interest as an instance of essential hypertension, probably well established long before pregnancy whilst the renal function had remained intact. Such a condition needed very close watching in hospital, so that the renal function could be tested at any sign of rising pressure. Continuation of the pregnancy was only permissible while the blood pressure remained unaltered and no signs of renal involvement appeared. A second pregnancy was contraindicated.

Skiagrams.

DR. D. G. MAITLAND showed a series of lantern slides of X ray films illustrating: (i) early pregnancy, showing the various types of presentations and multiple pregnancies; (ii) the various degrees of pyelitis of pregnancy, demonstrated by intravenous pyelography; (iii) congenital atresia of the jejunum and the diagnosis of intrauterine fœtal deformities; and (iv) pulmonary tuberculosis associated with pregnancy.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Moore, Geoffrey Henry, M.B., B.S., 1936 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on Wednesday, September 9, 1936, the President, DR. C. H. OSBORN, in the chair.

Hæmolytic Icterus with Splenomegaly.

DR. IAN WOOD showed a girl, six years and six months of age, who had been sallow and pale during the first and second years; she had not been severely jaundiced in early infancy. She had been subject to colds and attacks of influenza, and had become paler, with a slight icteric tinge. The tonsils were removed in the latter part of 1935, and in the following December she had an attack of vomiting with very definite jaundice, though the stools remained dark. She was admitted to hospital in Adelaide, and was found to be anæmic as well; the red cells numbered 3,500,000 per cubic millimetre, and the colour index was 0·8; a positive indirect Van den Bergh reaction was obtained; the corpuscular fragility was said to be normal, and the spleen was palpable as far down as three fingers' breadth below the costal margin. She came over to Melbourne, and her condition was reinvestigated. The spleen appeared to be about the size indicated, the red cells numbered 4,000,000 per cubic millimetre, and the colour index was 1·0; she had had iron therapy in the meantime. Some variation was noted in the shape and in the size of the red cells, and there was a lot of polychromasia; the percentage of reticulocytes was high and was estimated to be between 10 and 15; both series of white blood cells were of normal appearance. Dr. Reginald Webster had carried out a fragility test and had demonstrated very definitely that the fragility was increased; the cells were hæmolysed by a 0·6% solution of salt. The child still had a big spleen, but in other members of the family the spleens were not palpable. Her father was anæmic when young, and so was his sister, but there had been no history of jaundice. The child's blood serum failed to yield the Wassermann reaction. Dr. Wood asked for an expression of opinion concerning the advisability of splenectomy.

DR. J. W. GRIEVE said that he thought splenectomy was indicated and that the results in this disease were remarkably satisfactory.

DR. REGINALD WEBSTER said that there were certain conditions other than hæmolytic jaundice which led to increased corpuscular fragility, but that he had carried out the test with a series of dilutions varying by only 0·025% to obviate the possibility of an error in dilution, and all the tubes had been hæmolysed from 0·3% to 0·6% inclusive. This amount of increased fragility was pathognomonic of familial hæmolytic jaundice. Dr. S. O. Cowen had said that the fragility remained unaffected after splenectomy, but others had stated that it returned to normal.

Gangrene Following Purpura.

DR. D. O. BROWN showed a boy, two years and three months of age, whose legs he had amputated below the knees on account of *purpura fulminans* with rapid gangrene. Dr. N. Cust, resident medical officer, had seen the child on admission on the day after the hæmorrhages had commenced, and had established the fact that a temporary thrombocytopenia had been present. (It is proposed to publish a full report of the case at a later date.)

DR. H. C. MCLORINAN said that it was doubtful whether the *purpura fulminans* had complicated mild scarlet fever, although a contact had developed mild scarlet fever on the day before this child was admitted to the Children's Hospital. At the Queen's Memorial Infectious Diseases Hospital, over a period of twenty years, covering approximately twenty thousand cases of scarlet fever, there had been only one case of *purpura fulminans*, which had occurred in the fourth week of illness, and the patient had died after a few days; this incidence was not more common than in the general population, and it did not appear certain that the complication could be regarded as a complication of scarlet fever. The child had not had any symptoms of streptococcal toxæmia.

DR. S. W. WILLIAMS, medical superintendent, commented on the rarity of the case. He believed that there were only twenty-five cases of gangrene following a purpuric hæmorrhage, of which only seven had been bilateral. Only three of the patients had survived, and he thought that Dr. Brown's patient could be added as the fourth.

DR. R. WETTENHALL referred to the rapid and miraculous check which might follow treatment of an extravasation of blood in purpura by injecting whole blood, possibly from a parent. He also mentioned underlying pellagra as the cause of dramatic fatal occurrences from sudden bleeding.

Bronchiectasis.

DR. A. P. DERHAM showed a child, two years and three months of age, who, after several attacks of pneumonia, had been shown by the use of lipiodol to have a very definite bronchiectasis. Dr. Derham had shown the child at this stage because it was proposed that lobectomy should be performed at an early date. It was hoped that the child would be shown again later.

Transverse Myelitis.

DR. J. W. GRIEVE showed a girl, twelve years and six months of age, who had been admitted to the Children's Hospital on August 4, 1936, and he referred to the clinical history of a boy, thirteen years and nine months of age, who had been admitted on May 27, 1936. The two histories were remarkably similar. The boy had been awakened in the night twenty-three hours before admission by a pain in the right hip and toe. He had not previously had any illness of any importance. Two hours later he had experienced pain in the right hand, and the fingers felt numb; these alterations of sensation spread up the lateral aspect of the right arm, across the mid-scapular region and down the left arm, and the pains kept him awake. Difficulty in breathing occurred and persisted, and he developed weakness in the right side of the body and was unable to walk; he fell over on getting out of bed. There was no loss of sphincteric control, and apart from a minor degree of weakness of the lower portion of the right side of the face, the cranial nerves were unaffected. The triceps muscle was powerless and flaccid, and no triceps jerk could be elicited; there was gross weakness of the flexors and extensors of the wrists and fingers, the right side being affected more than the left; the biceps muscles were weak and the biceps jerks were obtained with difficulty. The muscles of the shoulder girdle appeared to be normal, but there was gross weakness of the muscles of the back; the intercostal muscles and the *recti abdominis* were not normal. There was great weakness of the right lower limb and a little of the left. The right plantar reflex was regularly extensor, but the response was equivocal on the left side; the right superficial abdominal reflex could not be elicited. The pyramidal tract was impaired, involving the right lower limb. Two or three days later impairment of sensation of pain, heat and cold was demonstrable on the whole of the left side up to a point two inches below the clavicle in the third or fourth dorsal segment, and the impairment extended into the left upper limb. The right side was similarly affected between the groin and the level two inches below the clavicle. The posterior columns appeared to have escaped; tactile and deep sensations were normal. The segmental distribution corresponded with an upper limit at the fifth cervical segment and extended down to the lowest thoracic segmental level. On a number of

occasions the cerebro-spinal fluid was found to be under normal pressure, and the estimations of the number of cells and of the globulin content and of the chlorides present were established to be within normal limits. No abnormality was discernible in the skiagram of the cervical spine and the optic fundi were also examined and found to be of normal appearance. A Queckenstedt test of the cerebro-spinal fluid gave no reaction, and the blood serum and cerebro-spinal fluid failed to yield the Wassermann reaction. There was a very slight rise of temperature at the onset and for three or four days, during which the patient was somewhat drowsy and emotional, but in other ways the general condition had been very good. Little improvement in the upper limbs occurred for three or four weeks, but later the boy had recovered very good power in each arm, though there was some residual weakness of both triceps muscles. The lower limbs and trunk muscles had improved more rapidly; he was able to walk about, though the right leg was a little spastic. Right ankle clonus had appeared about one month after the child's admission to hospital, and the jerks had remained hyperactive and the right plantar reflex was extensor. Sensory impairment was still detectable over the same area, but it was not so obvious as it had been.

The girl who was shown by Dr. Grieve had not had any previous serious illnesses and had six healthy brothers and sisters, though her father had died from a chest condition which might have been tuberculosis. She awakened at 1 a.m., fifteen hours before her admission to hospital, with pain in the forearm on the radial side and on the outer aspect of the arms, which she described as an aching type of pain; paræsthesia was also present in both sides. Forty-five minutes later, on getting out of bed, the patient found that her arms and legs were weak. She went back to bed and was able to sleep, but next morning, seven hours after the onset, the weakness in the arms and legs was found to be very much worse. On the first occasion after the onset of pain she had been unable to pass urine, but no further sphincteric disability had occurred.

When she was examined after admission to hospital, no disturbance of the cranial nerves was found, but gross weakness of both upper extremities was present; she was unable to adduct the arms or to move the fingers; the wrists were weak and the triceps and pectorals were flaccid; the back muscles, the intercostals and the recti were weak, as were the legs, especially the right. The triceps jerks were absent, but both the biceps jerks were active; the knee jerk was much more active on the right side than on the left; the right plantar reflex was extensor with flexor withdrawal response, and the left equivocal; the superficial abdominal reflexes were absent from both sides. Sensory impairment of pain, heat, and cold sensations were defined over the body up to the level of a line three inches below the clavicle and over both upper extremities, except for a band in the sixth cervical segmental distribution area. Tactile and deep sensations were normal. The affected segments were estimated to range from the seventh cervical level down to the last thoracic segment. The cerebro-spinal fluid pressure was measured to be 220 millimetres of water; the cell counts and estimation of chloride were within normal limits, though there was a slight increase in globulin content; but the Queckenstedt reaction was normal.

The course of the condition had been similar to that described in the case of the boy. The left arm had improved considerably and the right slightly; the right leg had improved rapidly and much more quickly than in the former case, but the sensory changes were similar. The left pupil had become larger than the right, but both reacted briskly to light.

Dr. Grieve commented on the rarity of transverse myelitis in children and of the interest attaching to two such similar histories. He believed that one or two other very similar cases had occurred at the hospital recently, and invited a discussion on the possible ætiology of the infection; he considered that it was probably atypical poliomyelitis.

DR. GRAEME ROBERTSON said that Dr. Grieve had been kind enough to afford him the opportunity of seeing the two patients at an early stage of their illnesses, and, in addition to providing him with a very pretty exercise in anatomical localization, they had given him much thought as to the ætiology. He had little doubt that cases of myelitis were more common in children than was generally recognized, escaping notice in Australia under the generic term of poliomyelitis. Such a case had come under his notice earlier in the week: a child, fifteen years of age, had been diagnosed as suffering from poliomyelitis and had been treated with convalescent serum. She gave a history similar to each of these patients, but on examination there was found to be much less involvement of lower motor neurones and more complete interruption of pyramidal, extrapyramidal and sensory fibres at the fifth cervical segment—a transverse myelitis in the true sense of the word. Dr. Grieve's patients could, with much more justification, be labelled as suffering from poliomyelitis, for the incidence was certainly greatest upon the cells of the anterior horns of the cord, yet the signs presented did not quite fit in with the modern pathological conception of poliomyelitis. The infection, as had been shown by experimental work, probably entered the body via the olfactory nerve terminals in the nasal mucous membrane and travelled down the spinal cord, apparently by the pyramidal tracts, to the neurones in the anterior horns of the grey matter. There was reason to believe that an acute infection might by spread and œdema cause a transverse myelitis; but in all cases in the acute stage one would expect to find cells in the cerebro-spinal fluid owing to outpouring from the Virchow-Robin spaces, this having its pathological correlate in the intense cellular perivascular cuffing always evident in the acute stages. These patients were subjected to lumbar puncture several times, and first within twenty-four hours of the onset; in each case there were no cells in the cerebro-spinal fluid.

Dr. Robertson pointed out that the same difficulty applied in any other infective explanation, including the virus infections, and said that it was strange that these two patients, with the upper level of the lesion in the sixth or seventh cervical segments, involvement of the decussating fibres subserving pain and temperature over a somewhat similar extent to that of the anterior horn cells and of the pyramidal and spinothalamic tracts on the right side of the spinal cord, should each have had normal cerebro-spinal fluid. It suggested perhaps a clinical picture not yet understood.

DR. ROBERT SOUTHBY said that at the onset the condition had been hemiplegic in distribution, and in two or three days more extensive paralysis had occurred. He considered that these findings were consistent with the presence of normal cerebro-spinal fluid. The increased cell count in poliomyelitis was helpful if its presence was ascertained before weakness developed. Dr. Southby had seen fulminating cases with acute transverse myelitis and rapid ascent with fatal termination in three or four days without cells in the cerebro-spinal fluid, but the patients had been grossly paralysed when he had seen them. In the girl shown the wasted pectorals helped to confirm Dr. Grieve's view that the condition was atypical poliomyelitis.

(To be continued.)

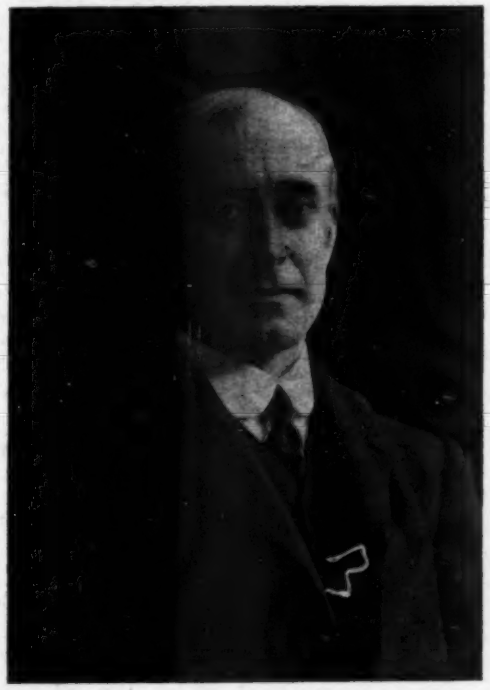
Obituary.

JAMES LINKLATER THOMSON ISBISTER.

WE are indebted to a colleague who wishes to remain anonymous for the following appreciation of the late Dr. James Linklater Thomson Isbister.

On November 30, at his home in Wollstonecraft, at the comparatively early age of sixty-seven, died James Linklater Thomson Isbister, after a painful and distressful illness of some weeks. I did not know him as a boy, but gather that his early school days were spent at the Grammar School at Ayr, and that he finished his schooling at Saint Peter's in Adelaide. From there he passed to the University of Adelaide, gaining a B.Sc., going through

the medical school, and finishing with the degrees of M.B., Ch.M. He obtained an appointment at the Sydney Hospital, serving one year as a resident and two as pathologist. I did not know him at that time, but have a very clear recollection of his succeeding Dr. Kyngdon and taking up his general practice at North Sydney in 1898. I had myself started practice in the neighbourhood some months before. He made the usual ethical call on me, as a matter of courtesy—the ceremonial call which I am afraid is not so punctiliously made by the present generation. He struck me then as a rather reserved, somewhat shy and diffident young man, rather apologetic in his manner for his "intrusion", but withal very courteous in his demeanour, and I liked him well. His progress in his practice was phenomenal, and before many years he was almost submerged by the demands made on him by his many patients, both private and "lodge". We worked together a good deal, and I was soon impressed by his absolute conscientiousness, his devotion to his patients' welfare, his high ethical ideals and his utter disregard for himself. His enthusiasm was inspiring, and he gave of his very best to every patient, no matter how humble, with apparently very little regard to the financial side of his work and his own pecuniary interests. His unceasing and unflagging energy in his work, the amount of time he gave to his cases with no apparent thought of his rest or his meals, used to fill me with amazement; and all his colleagues, I amongst them, found him impossible to live up to. No wonder he was so admired, loved and adored by his patients, young and old, and of both sexes! Some of his friends used to plead with him to let up in his work; but with no avail. The exhausting nature of his work was, of course, increased by the fact that he was all the time carrying out his duties as an honorary surgeon at the Royal North Shore Hospital. The inevitable breakdown came in 1908, when insomnia and some physical ailments compelled him to leave his work for a year or so, which time he spent in London, Scotland and at the home of his forbears, the Hebrides.



He came back and resumed his work, now in partnership with Dr. Throsby, also resuming his work as an honorary at the Royal North Shore Hospital, confining his attention latterly to gynæcology. He retired from the honorary staff of the Royal North Shore Hospital in 1928, a dissatisfied and disillusioned man, two others of the senior staff retiring at the same time, in sympathy and agreement with him. From that time on his career was entirely as a consultant in Macquarie Street.

Thus in brief terms may be stated his career, as I knew it, and I can give but a very inadequate idea of the excellence of his work—as a general practitioner, as a pathologist, as a surgeon, and as a gynæcologist. It was all of a very high order, and if the old definition of "genius" as an infinite capacity for taking pains be true, then Isbister was a "genius" of a very high degree; for in nothing was he more remarkable than for the extraordinarily meticulous, patient care for every detail in his work, leaving nothing to chance and thinking of every possible means to assure a correct diagnosis, and seeking for the employment of every therapeutic means for the benefit of his patients. These habits and methods caused him to work and worry excessively, and no doubt were the causes of his frequent insomnia and general poor health. But he was absolutely unalterable in his ways, and no expostulation from his friends could make him budge in the slightest from the course he had made up his mind to pursue. I know of no person to whom the term "impulsive" could be less accurately applied. He seemed to me in every clinical sense—indeed in any doing or action in his ordinary life—to think over every possible avenue of action carefully and meticulously, and I am quite sure it was quite alien from his nature to do anything on the spur of the moment. He was the source of great amusement to me that he would frequently make an appointment with me and ask for my opinion on some course of action he proposed to follow, in professional, business, personal or private matters. I frequently rejoined that I should be pleased to give my opinion, but that I was perfectly aware that nothing that I could say was in the slightest degree likely to cause him to alter the decision that he had already come to! And I have no doubt such was the experience of many others. For his fellows and colleagues he had many likes and dislikes; he was slow to make up his mind, but when he did like a man (or woman) his liking for, admiration for, and trust in this man (or woman) would be complete; if, on the other hand, he disliked a person (and sometimes it must be confessed that he formed an unfavourable opinion for reasons that were not easily understood by others) then his dislike was very complete and thorough. His views in some cases seemed to be somewhat narrow and unbendingly strait-laced, possibly a trait of character derived from Calvinistic forefathers. Many of his colleagues—in fact most of them—admired him, some did not care for him and seemed afraid of him, some of us were at times impatient with his manner and methods; but all were absolutely assured of his absolute probity and rectitude, his conscientiousness and desire to do the utmost that was in him for those under his care. He was not what is commonly known as a "good mixer". He was rather shy and diffident; and his apparently austere manner and "correctness" seemed to many to indicate that he had very little time for the levity, persiflage, badinage and trifling back-chat of repartee that takes up so much of the ordinary intercourse between men when not on serious business bent. He seemed to many to carry the theory of "scorning delights" too far, and so many were not at ease with him; they felt that he was too engrossed in "living laborious days" to take much enjoyment from the more trivial things of life. In this, I think, they were in error, because as a matter of fact he had a keen sense of humour and fun, and thoroughly enjoyed a humorous quip or *bon mot*. His was indeed a unique character: there was nothing shallow or superficial about him, and the longer one knew him, the better was he appreciated and admired. He lived a rather secluded and restricted social life, and shunned and abhorred any idea or suggestion of publicity. He was thoroughly serious in his duties during the years that he was on the British Medical Association Council, and he did a great deal of unostentatious work in connexion with his church and the church societies; but I am sure that the happiest time of his life was at his home, with his garden, his music and the companionship of his family circle. He was, I know personally, bitterly disappointed when, after much self-examination, he decided that it was quite impossible for him to offer his help on active service during the Great War; he thought he was temperamentally,

physically and nervously unsuited for such work; and I am sure he was right. But the work he did for, and the help he gave to, returned men and their families, both professional and financial help, was very great indeed. He did this almost by stealth and would have been greatly abashed and annoyed if he thought anybody knew of it. And the work he did as a matter of course for needy or sick members of his own profession and their relatives, to the nursing profession, for the Salvation Army, and for multitudes of others was very great indeed—and all with an utter lack of ostentation. I know from my own observation that his home life was a perfect one—there was absolute devotion to his wife, three sons and one daughter, a devotion that was perfectly mutual.

And so has left us a splendid doctor, a good citizen, a sincere, loyal and dependable friend, a devoted, loving husband, and a wonderful father. He was indeed "a veray parfit gentil knight". The profession and the world are the poorer for his passing.

"An Old Friend" has forwarded the following appreciation:

In the profession of medicine, in Sydney, we have had and still have men of many types and diverse abilities. We know the pure worldling—the medical tradesman—whose whole mental horizon, and all within its circle, is just the seeing of plenty patients and getting paid for so doing. Then we have the man who has a wholesome pride in continuing, ever and always, a student, and so doing his work well, with that laudable pride in his own skill and craftsmanship which makes the daily exercise of all skilled work a fine uplifting pursuit. Next we have, more rarely, the quite unworldly worker, who is content to live laborious days, on small pay, with just the joy of discovery or the pleasure of special work well done to reward him. These types of men we all know; some of them we like and some of them we admire and hold in high professional esteem. More than a few, unhappily, belong to the first category of whom I spoke, and in our inner sinful hearts, in moments of clearness, we sometimes recognize ourselves, and anyway we do not regard them as examples to be followed.

But there are yet others, few indeed perhaps, where, with professional excellence, with integrity and yet with a quite proper and proportionate regard for medicine as a means of earning a livelihood, in whom we find the higher qualities of mind and heart. These men we know to be transparently honest, even when we differ from them. Their word is their bond, they are generous and upright and wholesome in thought and life. There be, perhaps, few amongst us who rise to such high places—"stern peaks that dare the stars"—but certainly of that radiant company was James Linklater Isbister.

With his passing many of us feel that we have lost a dear comrade—a good exemplar of how to live, not for self alone—and a fine instance of a learned, all-round doctor and a safe and competent gynæcologist. In addition he was a man of light and learning, not only in the ancillary sciences of medicine, but also in general literature.

As I who write remember countless talks with the man, I cannot recollect ever leaving his presence without having gained some new knowledge, but, above all, with the consciousness of having held converse with someone very much better than myself.

Jimmie Isbister was all that I have said just now, and more; but, thank goodness, he was also very human and not a tepid angelic personality. He was quaintly alive to his own imperfections, and they were much fewer than he thought himself; and he also was able to see through the chinks in the armour of unrighteousness in others, so that he had at least a fair knowledge of the world and so was able to make a success of his career. A success deserved, got without pushing or window-dressing or humbug. He was painfully honest and meticulously anxious always to do the best, the very best possible for each patient. I believe his attitude to each of them was like that of Lister, as if they had come specially recommended to his care by God Almighty. It is a good spirit to have in one's heart in the performance of our high mission to the sick and sad and hurt.

I leave it to others to tell those of our profession who did not know him and his work at first hand where he served the public and how he did it. For myself, it is a satisfaction to tell them what a fine life Isbister led both in public and private, and what an inspiration to unworldliness and well-doing his example has been, and to record here our sense of personal loss in the passing of this much loved doctor and skilful surgeon.

Dr. Burton Bradley writes:

May I be permitted to express the sorrow and sense of loss that will be felt by the colleagues, friends and patients and Jimmy Isbister at his passing?

I knew him first as the respected physician of our family, later as a senior colleague, whose kindly advice and help were so freely given. I knew him, too, from the stories about him that were circulated in North Sydney in my boyhood and youth—such stories as would grow up around the beloved physician that he was. Maybe they were not always strictly true, maybe they contained a reflection of that kindly humour that ever played around his mobile mouth; but they all enshrined the ideal that he had built in many hearts.

I know of a little old woman, poor, lonely and very sick, whom Jimmy tended with loving kindness. He watched over her constantly, fearing that she might awake, perhaps dying, all alone.

Dr. Isbister graduated from Adelaide University, B.Sc. in 1891, and M.B., B.S. in 1896. He was a foundation member of the Royal Australasian College of Surgeons. From 1897 to 1898 he was a resident medical officer at Sydney Hospital, and from 1898 to 1899 resident pathologist. He was for many years consultant gynæcological surgeon at the Royal North Shore Hospital of Sydney. He practised at North Sydney for many years and later became a trusted consultant in Macquarie Street.

Dr. Isbister leaves behind a widow, three sons and a daughter, all fired with a desire to live worthily and to play their part in what he considered to be life's vast purpose.

Correspondence.

HIGHER DEGREES AND COUNTRY HOSPITALS.

SIR: It is no doubt desirable that, other things being equal, preference should be given to those with higher degrees when making appointments to honorary positions at country hospitals, but that after the lapse of five years higher degrees should be obligatory is quite another matter.

Just as educationalists are becoming dissatisfied with examinations and are trying to minimize their importance and their number, the surgeons are trying to increase both!

Academic distinctions certainly prove academic ability, and moreover that much reading and thought have been given to improving one's qualifications and thus give one a good start in life, but it is certain that character, temperament, personality, probity, manner, reputation, judgement, dexterity, results, count far more both with patients and with *confrères*. Laymen do not need to read Sir Frederick Treves's story of "The Idol with the Hands of Clay" to know that experience of the world and of practice are of infinitely more importance than higher degrees.

A man or woman may be an admirable thinker or doer and yet a poor examinee; the man who thinks with his hands may be a marvellous surgeon and yet have a struggle to get even his baccalaureate, and thus be for ever frustrated in his ambitions should higher degrees become obligatory.

Sir Alan Newton regards the newly qualified bachelor of surgery as fit only to suture a cut or open a boil, but surely he does not here do justice to the teaching of his own medical school and to the quality of its graduates.

Bachelors, like masters (on Sir Alan's own showing), require experience, and they are wise enough to get it, and if wiser still go on learning all their lives, even though they never do another examination!

In the country it is practically impossible to specialize in surgery, and it is likewise impossible to avoid surgery. The record of country surgeons (most of them bachelors, at least academically) is amazingly good, judged by the esteem which they inspire in their patients and their fellow practitioners. Why after five years should they have to yield place to less experienced juniors who hold a higher degree?

When drastic changes are made in organization (as, for instance, when professions are first registered) all existing rights are conserved, the new conditions applying to the newcomer. This is the accepted ethical custom, and should not be forgotten either by the College of Surgeons or the Charities Board of Victoria. If preference be given to holders of higher degrees, other things being equal, eventually (not in five years) most honoraries will possess such degrees should experience prove them desirable.

Yours, etc.,

MARY C. DE GARIS.

Gable House,
268, Latrobe Terrace,
Geelong, Victoria,
January 11, 1937.

THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

SIR: The Council of the Medical Benevolent Association of New South Wales wishes to convey its thanks to all those who so generously contributed to the Christmas appeal issued in December.

The total sum collected amounted to £178 8s. 6d., of which £95 was immediately distributed to ten deserving members, and the balance was placed to the credit of the general fund.

Yours, etc.,

J. M. GILL,
Honorary Secretary.

135, Macquarie Street,
Sydney,
January 12, 1937.

"ENSOL."

SIR: It seems strange that Dr. Molesworth should make it his business to demand a complete trial and proof of the therapeutic and clinical value of "Ensol" when his own "Pandora" has been a tolerated series of experimental appliances during the past twenty years, and still is of doubtful therapeutical value in the complete treatment of cancer.

Since Dr. Molesworth lays such serious stress on the value of his challenge, namely, three selected cases, it may be fair to consider two cases selected at random, but of opportune time; these cases only recently I referred to an acknowledged specialist of deep X ray therapy, as follows:

Case I.—A primary squamous-celled carcinoma of the floor of the mouth, Grade IV (pathologist's report). The growth involved the alveolar margin, the right anterior pillar, and the lingual aspect of the jaw and the floor of the mouth and the side of the tongue. Treatment: Being surgically inoperable, X ray therapy was advised and carried out; the growth thrived under the X rays' influence; radium was considered, but our learned colleague considered the case insensitive to therapy, and the case was returned to me, no better, the growth much advanced, the outlook hopeless. I proposed "Ensol" to the patient, and after twenty-one days' treatment there is some local improvement.

Case II.—Recurrence: After removal of the primary scirrhous growth fourteen years previously; at the site of a stab wound; fourteen months ago a tumour involving the skin was widely excised and readily healed. To make sure, the case was referred for X ray examination and therapy. Several treatments have been given during the past fourteen months, until I observed that a large, diffuse, fixed mass had made its appearance; a small fungating nodule presented in the axilla, and the skin for four inches over the anterior axillary aspect became red, thickened and indurated, not unlike cancer "*en cuirasse*". After pointing out this condition it was proposed to implant radium, which the patient did not allow. I then proposed "Ensol" to this patient, and treatment has been carried out for four weeks with very obvious regression and improvement of the clinical condition. Both these cases are capable of being observed accurately.

Surely the founders of "Ensol" are just as much entitled to use their product in such cases as the protagonists of X rays and radium. There is this difference in these two cases: X ray therapy has failed, though the "Ensol" treatment, whilst encouraging, is too incomplete to be evaluated. One must remember that X rays have had the field, heaps of money, the backing of a section of the profession, every method of research, for the past twenty years. Has it justified itself? Do 30% of all cases result in cure?

The history of the great reforms of medicine and surgery is full of instances where the sponsors of reform have been retarded in every way, and if "Ensol" can be developed to assist in the conquest of cancer, and X rays disappear from our therapy, great will be the day. However, it ill becomes any of our profession to stifle original work when seeking a cure of cancer, which so far has baffled us all.

Yours, etc.,

HARRY R. SCRIVENER.

Gordon,
New South Wales,
January 17, 1937.

INTERNATIONAL CONGRESS ON MENTAL HYGIENE.

SIR: I have been asked to bring to your notice the forthcoming meeting of the second International Congress on Mental Hygiene, which will be held in Paris from July 19 to 23, 1937. Interested organizations and individuals are invited to participate in this congress, the subscription to which has been fixed by the National League of Mental Hygiene at 500 francs (French). The Secretary-General of the Congress is Dr. Georges Genil-Perrin, of 1, Rue Cabanis, Paris, XIVe.

Yours, etc.,

J. H. L. CUMPSTON,
Director-General of Health.

Department of Health,
Canberra,
January 13, 1937.

Books Received.

DISEASES OF THE NAILS, by V. Pardo-Castello, M.D., with foreword by H. Fox, M.D.; 1936. London: Baillière, Tindall and Cox. Royal 8vo, pp. 199, with illustrations. Price: 16s. net.

THE CLINICAL USE OF DIGITALIS, by D. Luten, A.B., M.D.; 1936. London: Baillière, Tindall and Cox. Royal 8vo, pp. 246. Price: 16s. net.

A THEORY OF CANCER AND THE PRACTITIONER AND MNEMOTHERAPY, by D. Roosen, M.D.; English translation revised by C. F. Marshall, M.D., F.R.C.S.; 1936. London: Baillière, Tindall and Cox. Crown 8vo, pp. 81. Price: 3s. 6d. net.

APPLIED DIETETICS: THE PLANNING AND TEACHING OF NORMAL AND THERAPEUTIC DIETS, by F. Stern; 1936. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 285, with 52 tables. Price: 16s. net.

Corrigendum.

OUR attention has been drawn to a misprint that occurred in the journal of January 16, 1937, at page 113. In the discussion at Adelaide, Dr. Gilbert Brown is reported as having stated that seven ounces of paraldehyde were given at the Liverpool Maternity Hospital in seven ounces of olive oil by the rectum. "Seven ounces" of paraldehyde should read "seven drachms".

Diary for the Month.

JAN. 25.—Victorian Branch, B.M.A.: Council.
JAN. 27.—Federal Council, B.M.A.: Half-Yearly Meeting (Melbourne).

Medical Appointments.

Dr. R. Wallace has been appointed Government Medical Officer at Rosewood, Queensland.

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Dr. R. F. Dilger has been appointed Government Medical Officer at Yenda, New South Wales.

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Dr. R. Williams has been appointed Government Medical Officer at Southport, Queensland, and a Health Officer under *The Health Acts*, 1900 *to* 1934, of Queensland.

* * *

Dr. G. J. Williams has been appointed Honorary Ophthalmic Surgeon at the Newington State Hospital and Home, New South Wales.

* * *

Dr. S. Richards has been appointed a member of the medical board constituted under the *Workmen's Compensation (Lead Poisoning—Broken Hill) Act*, 1922, of New South Wales.

* * *

Dr. C. A. Finlayson and Dr. J. S. Proctor have been reappointed Official Visitors at the Mental Hospital at Parkside, Victoria, pursuant to the provisions of the *Mental Defectives Act*, 1935, of Victoria.

* * *

Dr. F. J. Donoghue has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, of Victoria, Certifying Medical Practitioner at Leongatha, Victoria.

* * *

Dr. D. G. May has been appointed Certifying Medical Practitioner at Koondrook, Victoria, in accordance with the provisions of the *Workers' Compensation Act*, 1928, of Victoria.

* * *

The following appointments have been made at the Adelaide Hospital: Second-Year Resident Medical Officers, Dr. W. H. Baudinet, Dr. O. W. Leitch, Dr. T. D. Kelly, Dr. C. S. Swan; First-Year Resident Medical Officers, Dr. Betty Ambrose, Dr. C. F. Chapple, Dr. J. F. Frayne, Dr. K. H. Heard, Dr. K. W. Hodby, Dr. J. Ray, Dr. W. F. Salter, Dr. E. J. Tamblyn, Dr. G. M. Watson, Dr. F. E. Welch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix, xx.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Clinical Assistant to Honorary Radiologist.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Officers.
THE PUBLIC SERVICE BOARD, NEW SOUTH WALES: Medical Officer (Male).
THE RYDE DISTRICT SOLDIERS' MEMORIAL HOSPITAL, RYDE, NEW SOUTH WALES: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 305, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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